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PRINCIPAL INVESTIGATOR: Mary Klein, Ph.D.

CONTRACTING ORGANIZATION: Albert Einstein Healthcare Network
Philadelphia, Pennsylvania 19141

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Mary Klein, Ph.D.

E-Mail: mklein@einstein.edu

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14. ABSTRACT

The goals of this project were: 1) to study the temporal relationship between activity level and health status in polio survivors and to compare the results with those obtained from an age-matched control population and 2) to look at the effect of localized muscle weakness and the associated compensation response on performance of a walking task. Simulation modeling techniques were used to identify factors critical to task performance, which provided valuable information for optimizing rehabilitation interventions for polio survivors and other populations with lower extremity muscle weakness. A total of 97 polio survivors and 116 controls were enrolled and tested for Study #1. Llongitudinal data was analyzed. For Study #2, the functional deficits database was compiled and an analysis was performed using the mechanical-based compensation scheme.

15. SUBJECT TERMS
polio survivors; physical activity; muscle weakness

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INTRODUCTION

Controversy exists regarding the relationship between strength, functional performance, and health status in the post-polio population, and the role of activity level in this relationship. Polio survivors tend to have a higher incidence of muscle and joint pain and higher levels of fatigue after normal daily activity compared to the general population. One possible explanation is that, because of their residual muscle weakness, polio survivors perform their activities at a higher activity intensity level than their peers, which may, in turn, make them more susceptible to musculoskeletal problems. However, there have not been any systematic studies performed to confirm this theory. Therefore, one of the goals of this project was to study the temporal relationship between activity level and symptoms of pain and fatigue in polio survivors and to compare the results with those obtained from a control population. A need also exists for improved neuromuscular, musculoskeletal, and link segment dynamic models of common daily activities that can be used to predict the compensatory strategies that will be employed when muscle weakness is present. Therefore, another goal of this research was to look at the effect of localized muscle weakness and the associated compensation response on performance of a walking task. Simulation modeling techniques were used to identify factors critical to task performance, which provided valuable information for future use in optimizing rehabilitation interventions for polio survivors and other populations with lower extremity muscle weakness.

BODY

Study #1: An Analysis of Health Status and Activity Level in Polio Survivors Over Time

Rationale:

Within the disablement process, there is an intuitive causal chain (e.g. pathology leads to decreased muscle strength which causes difficulties in daily living tasks). However, there may also be feedback loops involved in which physical activity plays an important role. Difficulties in performing tasks may cause decreased levels of physical activity that can lead to a further decline in strength and worsening of disability. Increased or high levels of physical activity can result in overuse injuries in either the muscles that are weakened from polio or in other muscles used to compensate for them. To date, little is known about the patterns of physical activity and the effects of physical activity level on musculoskeletal symptoms in older adults with physical impairments, particularly those with chronic conditions like post-polio syndrome.

Previous research has shown that muscle strength in polio survivors decreases at an accelerated rate compared to the rate of deterioration associated with normal aging.[1] Polio survivors also tend to have a higher incidence of muscle and joint pain and higher levels of fatigue after normal daily activity compared to strength-matched controls with no history of polio.[2] However, it is not clear whether level of activity affects the rate of strength loss and symptom development in polio survivors over time or whether deterioration occurs independent of activity level. Level of activity may influence physical function by effecting physiologic changes in strength and symptomatology or may merely be a marker for health status and physical function. Theoretically, there is an activity window that lies between complete inactivity and maximum vigor that will provide the benefits of loading, muscular contractions, and cardiac conditioning for polio survivors without the detrimental affects of overuse. Additional research is needed to help define this “activity window” for polio survivors and other

populations with disabilities. However, in order to begin to address this issue, more research is needed on the relationship between symptoms commonly associated with overuse (pain and fatigue) and physical activity patterns, and how these variables interact to affect disability in the post-polio population.

Subjects:

Polio survivors and controls were recruited from the community at large (including the surrounding four-state area: Pennsylvania, New Jersey, Delaware, and New York). Informed consent to participate in the study was obtained from all subjects. The study had prior approval from the Albert Einstein Healthcare Network's Institutional Review Board and the Department of the Army's Human Subjects Review Board before implementation.

Objectives:

1. To compare pain and fatigue ratings and activity frequency and intensity among polio survivors and controls.
2. To examine the factors related to activity level and symptom status over time.
3. To use longitudinal modeling techniques to examine the association between physical activity level, pain severity, fatigue level, and leg weakness in polio survivors and controls.

Method:

We recruited 97 polio survivors and 116 controls with a mild to moderate degree of lower extremity impairment to participate in this study. All subjects had to be able to ambulate on level surface with (or without) an assistive device. Control subjects must also have reported

difficulty in performing one or more of the physical activities listed in the physical function scale of the short-form health survey SF-36, which includes lifting or carrying groceries, climbing stairs, bending, kneeling, stooping, walking several blocks, and bathing or dressing oneself.

Exclusion criteria included recently diagnosed medical conditions like kidney failure requiring dialysis, rheumatoid arthritis, or recent cancer (other than skin) with ongoing treatment; uncontrolled hypertension; unstable angina pectoris or any other cardiac and/or respiratory conditions that are uncontrolled; uncontrolled seizures; any disease, illness, or injury that might affect muscle strength, such as uncontrolled diabetes, stroke, Parkinson's disease or other neuromuscular disorders other than polio; and any cognitive disorders that might impede the ability to understand what is involved in the study or the motivation to perform the strength tests.

Prior to any testing, subjects were asked to complete a subject history and polio history form (polio survivors only). The subject history form included basic medical history information, while the polio history form included information on date of original polio infection, limbs affected, etc. Height and weight were measured at each visit using a standard scale. Body mass index (BMI) was calculated using the following formula: BMI = weight (lb) x 705/ height (inches) / height (inches).

Activity Level: To obtain an objective measure of amount of walking activity, subjects were asked to wear a StepWatch activity monitor (SAM) for 5-7 days. The monitor was worn just above the left or right malleolus and was set to store the number of steps in 1-minute intervals. At the initial visit, the monitor was calibrated based on the individual's cadence and gait. The monitors were worn continuously for the 5-7 day period except during sleeping and water activities, such as bathing, showering, and swimming. Measures of the mean daily step

activity and number of steps at low (1-15 steps/min), medium (16-30 step/min) and high activity (>30 steps/min) levels were collected for analysis.

In addition, a measure of reserve walking capacity was obtained by calculating walking speed differential. Each subject was timed as they walked down a 10 m indoor walkway twice, once at a pace that was "normal and just right" for them and once as fast as they could. This test was performed at the initial visit and again approximately one week later when the activity monitors were returned. The walking speed differential was defined as the difference between maximum walking speed and the comfortable walking speed. When the activity monitor was returned, subjects were asked to give a subjective estimate of their activity level over the past week using the Physical Activity Scale for the Elderly (PASE).[3] This instrument dealt with occupational, household, and leisure activities taking place over the previous one-week period. An activity score was calculated from weights and frequency values.

Fatigue: The Fatigue Severity Scale (FSS) was used to measure level of fatigue during the activity monitoring period.[4] This scale was originally designed to measure the fatigue experienced by people with multiple sclerosis (MS), but has since been used to quantify effects of fatigue in patients with chronic fatigue syndrome (CFS) and post-polio syndrome (PPS).[5] The scale was designed to measure the severity of mental, physical and total fatigue by scoring responses to scale items. Subjects responded to each item using a 4-point rating scale, with higher numbers indicating more severe problems. The Fatigue Scale has been demonstrated to be a reliable and valid measure with high internal consistency.[4] Subjects were asked to rate their level of fatigue using this scale on a daily basis when they returned the activity monitor to the clinic. During the monitoring period, subjects were also asked to rate their fatigue at the end of each day on a scale from 0 – 10.

Pain: Subjects were asked to give daily pain ratings during the times that they were wearing the activity monitor. They identified the areas where they were experiencing muscle or joint pain and then for each area identified, they rated their pain severity on a scale from 0 - 10. An overall measure of daily pain severity was calculated by taking an average of the pain scores for the individual locations.

Strength: Voluntary maximal isometric strength was measured in the bilateral knee extensor muscle groups using a Microfet2 hand-held dynamometer. The subjects were given strong verbal encouragement during each trial. Initially, two trials were performed. If these two trials were not within 5% of each other, a further two attempts were allowed. The highest value recorded from each leg was taken as the definitive maximum contractile force (MVC). A review of the data from our previous studies involving polio survivors and a similar protocol for strength testing on the knee extensor muscle group revealed that fewer than 0.8% of the strength trials attempted for this muscle group were either invalid or missing due to pain.[6,7] Measures for overall bilateral knee extensor strength and strength in the weaker knee extensor were used for analysis.

All subjects returned to the Research Clinic for follow-up testing at approximately 3-4 month intervals over the two-and-a-half-year study period. The follow-up testing periods had the same protocol as the initial testing period.

Statistical Analysis:

Descriptive statistics were calculated, means and medians were closely examined and plots of each variable were inspected for asymmetry and long-tailedness and normality. When appropriate, transformations (e.g. log function) were made. Data were analyzed using the Stata

9 and S-PLUS 7 and software packages. All analyses controlled for within-subject correlations (clustering) from multiple observations on the same subject. Multivariable mixed models with random intercepts or both random intercepts and random slopes were used to assess the independent predictors of the study outcomes and, when possible, to compare the polio survivors with PPS, without PPS, and the control group. The models were adjusted for age at baseline, time in study, gender, initial body mass index (BMI), educational level, living alone or with other, season, and the report of the following conditions at baseline: osteoporosis and arthritis. Product interaction terms considered were between group and each of the following variables: age at entry, time in study and initial BMI. Interactions were retained in a model if $p<0.05$. In addition, the results of separate models for each group are reported (adjusted for strength of the weaker knee and the control variables listed above). Because the outcome variables were non-negative and right skewed, models using logarithmic transformations of the outcomes were also evaluated. Model fit was compared using the likelihood ratio test or the Aikaike Information Criteria (AIC). Pain and fatigue were analyzed using autoregressive mixed models in order to control for the previous day's values of those outcomes and the previous day's activity levels. Diagnostic plots, including residual plots and plots to assess the normality of both fixed effects (Q-Q plots) and random effects were evaluated for each multivariable model. Unless otherwise stated, p values are multivariable adjusted.

Results:

A total of 224 polio survivors and 322 adults with no history of polio were screened for this study. Of this number, 97 polio survivors and 116 control subjects were enrolled. Visit information is summarized in Table 1 and demographic information is listed in Table 2. One

polio survivor was enrolled in the study and then had to withdraw after the first visit due to a medical emergency unrelated to the study (bowel obstruction). Four control subjects were consented and enrolled in the study and then were dropped by the investigator due to complicated cognitive and medical issues that were identified during the review of their medical history.

Of the 96 polio survivors who completed the first set of testing sessions, 72% reported symptoms of post-polio syndrome (PPS). However, only 63% of those subjects had been diagnosed with PPS by a physician. The average number of years since the original polio infection was 57.76 (8.09) years for all the post-polio survivors in this study. When asked to rate their current health status on a scale from 0 (worst it could be) to 100 (best it could be), the average rating for the polio survivors was 74.64 (17.63). Twenty-eight percent (28%) of the polio survivors felt that their health status was improving overall, while 7% said their health status was decreasing. The remaining 65% reported no recent change in health. The majority of controls (79%) also reported that their health status remained stable, with little or no significant change. However, sixteen percent (16%) of the controls reported their health status was improving and 5% reported a recent decrease in health status, which resulted in average health rating of 78.83 (14.71).

Table 1. Number of Subjects Who Completed Each Set of Testing Sessions

Group	V1/2	V3/4	V5/6	V7/8	V9/10	V11/12	V13/14	V15/16
Polio survivors	96	80	69	59	45	35	29	29
Controls	113	93	85	77	61	56	42	27

Table 2. Baseline Characteristics- Mean (SD) or Percentage (%)

Baseline characteristic	Controls	Polio (not PPS)	Post-polio syndrome (PPS)
Number of subjects	112	31	65
Female	63%	52%	45%
Caucasian	77%	90%	92%
Age at baseline (years)	72.3 (9.3)	66.3 (8.5)	62.5 (9.0)
Initial body mass index (BMI)	27.5 (5.4)	26.3 (5.6)	27.2 (4.4)
overweight 25<BMI<30	38%	23%	35%
obese BMI $\geq$ 30	25%	23%	29%
Strength of weaker knee extensor (lb)	37.5 (17.8)	27.1(25.9)	19.6 (20.2)
Knee extensors- bilateral strength (lb)	81.5 (36.7)	66.6(48.8)	56.3 (37.5)
Maximum walking speed (ft/sec)	5.0 (1.0)	4.6 (1.4)	4.3 (1.3)
Normal walking speed (ft/sec)	3.6 (0.6)	3.4 (0.9)	3.2 (0.8)
Percent who use braces	0%	39%	48%
Percent who use assistive device	0%	26%	45%
Dizzy	14%	6%	20%
Osteoporosis	23%	23%	14%
Arthritis	31%	39%	31%
History of falls	27%	58%	71%
Live with other	64%	77%	83%
Completed college degree	41%	55%	57%

Activity Level

Analysis of the PASE data revealed that the association of age at baseline with PASE differed in the PPS and non-PPS groups (interaction statistically significant at $p = 0.03$). Thus no conclusion could be drawn regarding the multivariable adjusted comparisons of self-perceived activity in the three groups. In general, while the polio survivors with PPS had the lowest mean PASE score and the polio survivors without PPS had the highest, there was no statistically significant difference between the scores for each group.

When the analysis was run for each group separately, the results for the PPS group showed that subjects in this group perceived themselves to be less active as the strength of their weaker knee decreased ($p = 0.008$). In the non-PPS group, there was a trend for the PASE score to decrease with decreasing strength of the weaker knee, but this association was not statistically significant ($p = 0.09$). Subjects in the non-PPS group who reported osteoporosis had lower PASE scores ($p = 0.03$). In addition, older subjects in this group were more likely to have lower PAS scores than younger subjects ($p<0.001$). Inexplicably, we found that non-PPS subjects with arthritis had higher average PASE scores ($p=0.003$).

The results for the control subjects were similar to those in the PPS group in that average PASE score decreased as the strength of the weaker knee decreased ($p = 0.008$). Control subjects perceived themselves to be less active in winter than in other seasons ($p < 0.001$). Season was not a significant factor for either of the post-polio groups. Control subjects also perceived themselves to be less active as they aged ($p< 0.001$). There was no evidence of a change in PASE scores over time in either of the post-polio groups.

In the multivariable analysis for daily step activity, subjects in the PPS group had less daily step activity on average than subjects in the non-PPS and control groups ($p \leq 0.001$). Subjects in the non-PPS group averaged fewer steps than the controls ($p = 0.04$). On average, fewer steps were taken by subjects with osteoporosis and those with a higher BMI ($p \leq 0.004$). Subjects took fewer steps in winter than in spring or fall ($p < 0.001$). Overall, daily step count did not vary with age or gender.

Table 3. Means of activity level outcomes (across time) in controls, subjects with polio but not PPS and subjects with post-polio syndrome (PPS)

Activity outcome	Controls(1) N=111	p value (1) vs (2)	Polio (not PPS) (2) N=31	p value (2) vs (3)	Post-polio syndrome (3) (PPS) N=65	p value (1) vs (3)
Self reported activity score (PASE)	149	0.48 *	160	0.09 *	131	0.09 *
Number of steps per day	9462	0.14 (0.04)	8365	0.009 (0.001)	6248	<0.001 (<0.001)
Max-normal walking speed (ft/sec)	1.34	0.001 (0.001)	0.96	0.68 (0.59)	1.02	0.001 (<0.001)

* The top p values compared the means between the three groups in using unpaired t tests, taking clustering of observations within patients into consideration. The p values in parentheses (below) are from multivariable mixed models and controlled for age at study entry, time in study, gender, educational level, live with other, body mass index (BMI) at baseline, osteoporosis, arthritis and season. Asterisks (*) appear where statistically significant interactions between the group indicator variable and age at study entry exist because multivariable adjusted p values cannot be determined in that situation.

When daily step count was analyzed for each of the three groups separately, the results for the PPS group indicated that daily step count increased with strength of the weaker knee ($p < 0.001$). After controlling for knee strength, females in the PPS group took more steps than males ($p = 0.03$). Neither BMI nor osteoporosis were associated with daily step count in this group.

In the non-PPS group, although average daily step count increased with strength of the weaker knee, it did not reach statistical significance ($p = 0.13$). On average, non-PPS subjects with osteoporosis took fewer steps than subjects without osteoporosis ($p = 0.004$). However, no other variables were associated with daily step count in this group.

Among controls, the average daily step count increased with strength of the weaker knee ($p = 0.03$) and decreased as BMI ($p < 0.001$) and age at study entry ($p < 0.001$) increased. In

addition, the average daily step count was higher in spring and fall than in winter in the control group ($p \leq 0.003$).

Walking Speed Differential

As hypothesized, the average difference between the maximum and normal walking speeds, as measured in the laboratory, was lower among polio survivors than controls after multivariable adjustment (Table 3). However, PPS and non-PPS polio subjects had similar average walking speed differential values ($p= 0.59$). This walking speed differential (WSD) appeared to decrease as subjects aged because it decreased both with age at entry ($p=0.07$) and with time in study ($p=0.002$). On average, normal walking speed was closer to the maximum walking speed in those with osteoporosis ($p=0.02$).

Although WSD was greater in males ($p=0.03$) in the overall model, when the strength of the weaker knee was controlled for in the separate group models, gender was no longer associated with WSD in any of those models ($p>0.1$) but subjects with greater knee strength had greater average WSD in each group (all $p\leq0.003$). There were marginal declines in WSD with age in controls and subjects with PPS but such declines were not statistically significant in the non-PPS group ($p\geq0.15$). The walking speed differential decreased with increasing BMI in the subjects with PPS ($p = 0.01$), marginally in the control group ($p=0.08$), but not in the non-PPS group ($p=0.45$).

Pain Severity

It was hypothesized that the severity ratings for pain severity would be higher among polio survivors than controls. The bivariate analysis of the daily pain ratings showed a

significant difference in average pain severity between all three groups (all $p < 0.037$). Polio survivors with PPS had the highest average pain ratings and were more likely to report pain than controls or polio survivors who did not report symptoms associated with PPS (Table 4). Polio survivors without PPS were more likely to report pain than controls, but had the lowest average pain severity rating of all three groups.

A multivariable analysis of pain severity was run for each of the three groups. In addition to the standard variables listed in the analysis section above, the models included the number of low, medium and high steps from the previous day. The results for the polio group without PPS showed that women reported higher pain severity ratings than men ($p = 0.018$). There was a seasonal effect with higher pain severity ratings given in the spring and summer than in winter. In addition, the number of steps at high activity level during the previous day was also significant ($p = 0.009$) with subjects who took more steps at the high level reporting lower levels of pain severity.

For controls, initial body mass index was statistically significant ($p = 0.005$). Control subjects who were heavier reported greater pain severity. Season and number of steps at any level were not significant factors for this group. For the PPS group, none of the activity, strength or demographic variables were statistically significant predictors of pain severity.

Home Fatigue Ratings

The results for the bivariate analysis for home fatigue ratings showed a significant difference between all three groups (all $p < 0.002$). Similar to the results for pain, polio survivors with PPS were more likely to report fatigue and had the highest average fatigue ratings than controls or polio survivors without PPS (Table 4). Polio survivors without PPS had the

lowest average fatigue rating and were also the least likely to report any fatigue at the end of the day.

Since the home fatigue ratings were always done at the end of the day, we included the activity (low, medium and high steps) for the current day as well as for the previous day in the multivariable models. The results for the polio group without PPS showed that the number of high steps for the current day was marginally significant ($p = 0.059$) with the fatigue rating increasing with the number of high steps. However, none of the other variables were statistically significant.

The results for the control group indicated that the number of steps at both the high activity level ($p = 0.001$) and medium activity level ($p = 0.001$) for the current day, as well as the steps at each of these levels increased, the fatigue ratings also increased. Gender was also a significant variable for the control group ($p = 0.002$) with men reporting higher fatigue ratings, on average, than women. In addition, control subjects with more strength in their weaker knee extensor reported less fatigue than those control subjects with less strength in this muscle group ($p = 0.014$).

Activity level was also a significant factor for home fatigue ratings for the PPS group, with fatigue ratings increasing with the number of high steps on the current day ($p < 0.001$) and number of low steps for the current day ($p = 0.019$). Number of steps taken at the medium activity level for the current day was marginally significant ($p = 0.062$) with a trend for the fatigue ratings to increase as number of steps at the medium activity level increased. Steps from the previous day were not statistically significant at any level. Strength in the weaker knee extensor was marginally significant ($p = 0.053$) with a trend for fatigue ratings to be lower for subjects with more strength in their knee extensor.

Fatigue Severity Score (FSS)

For the polio group without PPS, strength in the weaker knee extensor ($p = 0.044$) and age at time of study enrollment ($p = 0.005$) were significant. Older subjects reported higher FSS scores than younger subjects in this group and subjects with stronger knee extensors reported lower FSS scores.

Within the control group, subjects with higher body mass index reported higher FSS scores ($p = 0.001$) and males reported higher FSS scores on average than females ($p = 0.015$). There was a trend for subjects with stronger knee extensors to report lower FSS scores, but this factor was not statistically significant ($p = 0.092$).

For the PPS group, subjects with a higher body mass index reported higher FSS scores ($p = 0.033$). In general, the FSS scores tended to decrease with time in study ($p = 0.042$). However, the amount of change was extremely small (coefficient: -.0001156).

Discussion

The results of this research describe the typical ambulatory activity of polio survivors and older adults with some degree of lower extremity impairment. Since the day-to-day variability in daily activity level, pain severity, and fatigue ratings for individual subjects was expected to be high, a repeated measures design was used in order to get a representative picture of activity patterns. Measurements were repeated every three to four months in order to capture any potential seasonal effects.

As hypothesized, the average number of steps per day, measured by the Stepwatch activity monitor, was lower in subjects with PPS than in controls and polio survivors without PPS. According to the previously published classification of pedometer walking activity[8], subjects in

Table 4. Pain, Fatigue and Activity Values (Mean (SD))

Outcome variable	Controls	Polio (not PPS)	Post-polio syndrome (PPS)
Number of subjects	112	31	65
Home Pain			
% reporting symptoms	80%	90%	94%
Ave. Pain severity	1.29 (0.2)	0.84 (0.2)	2.33 (0.2)
Home Fatigue			
% reporting symptoms	88%	84%	95%
Ave. Fatigue rating	2.39 (0.2)	1.20 (0.3)	3.57 (0.3)
Fatigue Severity Scale	25.87 (1.2)	24.64 (2.1)	38.68 (1.7)
Daily Step Activity			
Low	2938.7 (74.9)	2727.4 (171.8)	2331.2 (115.4)
Medium	3113.8 (131.6)	2821.0 (252.2)	2355.1 (165.0)
High	3409.3 (256.0)	2816.6 (449.4)	1590.2 (243.8)

the non-PPS and controls groups would be considered “somewhat active” on average, which means their daily activity is likely to include some exercise and walking. Subjects in the PPS group would be considered “low active”, which means their daily activity is not likely to include sports/exercise.

Subjects with PPS also perceived themselves to be less active than controls and polio survivors without PPS, and they averaged significantly fewer high activity steps per day than subjects in the other two groups. On average, subjects with PPS spent 11 minutes highly active per day compared to 38 minutes for controls and 23 minutes for polio survivors without PPS.

The walking speeds recorded for the non-PPS group in this study were comparable with those reported in a previous study on 24 patients with PPS.[9] However, the non-PPS and PPS groups in the current study were, on average, approximately 10 years older than the patients in the previous study. We had hypothesized that polio survivors tend to work at a level that is closer to their maximum capacity than controls. Both the normal and maximum walking speeds were lower, on average, in those with PPS than in controls and the difference between those speeds was smaller, which indicated that polio survivors with PPS have less reserve capacity. As a result, these polio survivors may be more likely to experience discomfort and fatigue with daily activity, which could predispose them to decrease their activity level over time. Our results indicated that polio survivors with PPS were much more likely to experience pain and fatigue and report higher severity ratings for pain and fatigue than controls or polio survivors without PPS. However, it is important to note that the results of this study did not show a significant decline in walking activity over the 2.5 year study period for any of the groups.

In general, seasonal changes in temperature, weather and day length are thought to modify habitual physical activity. Therefore, one might expect to see decreased levels of walking activity in the winter months due to the cold temperatures, shorter daylight hours, and potential for ice and/or snow. The results showed that season was a significant predictor of walking activity in the multivariable model with all three groups combined, with significantly less activity occurring in the winter months. However, when the analysis was repeated for each of the three groups separately and strength was added to the model, season was a significant factor for the controls only. The lack of significant change in daily activity levels among polio survivors between seasons may indicate that polio survivors have to maintain a relatively constant activity level just to accommodate their normal activities of daily living. Alternatively,

strength may be such a key factor for the post-polio groups that the variability between seasons was no longer significant.

In a previous study on walking activity in men and women over 65 years of age, age was negatively associated with walking speed, but not number of steps.[9] Older people were weaker and walked more slowly, but they did not walk less. In this study, the results were similar in that age was associated with the walking speed differential in the control group and PPS group, but was not associated with daily step activity.

Body mass index was an important factor in the control group and was inversely associated with daily step activity. As body mass index increased, there was also an increase in pain severity ratings and the FSS rating among controls. Body mass index was an important factor for walking speed differential and FSS rating among polio survivors with PPS. It was not significant factor for any of the activity or symptom variables for the polio survivors without PPS, which was also the group with the smallest percentage of people who were overweight or obese.

Activity level was a significant factor for predicting fatigue ratings in the polio survivors with PPS and control, with reported fatigue levels increasing as the number of steps, particularly at the high and medium levels, increased. There was a trend for a similar relationship among the polio survivors without PPS. One potential reason for lack of significance may be the smaller size of this group as compared to the other two groups. Number of steps at high activity level was a significant factor in predicting pain severity in polio survivors without PPS. However, none of the factors included in our model for polio survivors with PPS were significant. It is clear that further investigation is needed to determine the factors associated with pain severity in this population.

Study #2 Development of a Walking Model for Simulating the Effect of Localized Muscle Weakness

Executive Summary

Computer simulation of a forward dynamic model and human subject data were used to evaluate the potential of ipsilateral and contralateral joint moments to compensate for focal lower extremity weakness during walking. Using manual parameter tuning and mathematically based optimization techniques, the model was configured to track the stance leg joint angles of a representative human subject to an error of approximately 6.5 degrees RMS over an entire stance cycle. This represented a significant improvement of the model walking pattern over its originally published form.[10] The modified version of the model was acceptable for assessment of mechanical potential of compensatory responses.

The calculation of contributions of muscle groups to several key motions during walking was achieved and a graphical chart was compiled as a reference. It is included in the report results section. Furthermore, the potential for the various muscle groups to affect key motions during walking was also calculated and included in a similar graphical reference. These charts represent a movement towards objective assessment of the effects and potential effects of muscle action during walking. The basic methodologies and focus employed during this work have been proliferated since this proposal was written. As such, we have reviewed the small but significant body of work that has been published since we began this project. This body of work has helped to frame the results we present as well as allow the possible for a consensus on muscle actions to be developed. We are currently evaluating the data for trends and agreements.

The final objective of this work was to estimate how much mechanical potential accounts for and thus can predict compensatory response. This was evaluated in healthy normal subjects

in response to a focal short-term weakness. That weakness was produced by lidocaine injection. Data were successfully obtained for eight human subjects – no adverse reactions or events of any kind occurred. A mathematical method to estimate how joint torques in the body would change in response to that weakness was developed and coded into a computer program [Appendix III]. The math and subsequent solution was validated. Resulting joint torques were continuous and physiological in nature. Analysis of a single subject indicated that mechanical potential accounts for a small fraction of the observed compensatory response. After subsequent analysis, we can develop a meaningful quantitative estimate of how much mechanics may account for and thus help to predict compensatory strategies.

The current work has strong and potentially important ties to other projects sponsored by and overall goals of the Department of the Army. For example, the Virtual Soldier Research program at the University of Iowa aims to develop a virtual human model that can be used to evaluate equipment design and task performance on virtual prototypes before or instead of on actual military personnel. The project has developed a detailed computer model and has begun testing the model response. This model appears to generate movement predictions based on mathematical optimization algorithms. Neurophysiology is one area missing from the variety of fields incorporated to this model. This is a central element to our model and our preliminary evaluations have demonstrated that inclusion of these biologically inspired mechanisms and pathways increases a biomechanical model's ability to predict actual human responses. This is our ultimate goal – to be able to predict human responses to a variety of interventions that can lead to improved and more efficient delivery of clinical intervention. There may be considerable overlap and applicability of such a model to improving performance of the soldier in various military tasks as well.

1. Benchmarking & Sensitivity of Model Performance (Objective 1)

1.1. Summary

The purpose of this section of the work was to establish (benchmark) the baseline performance of our model and to understand how parameter variation affected that performance. The model, when suitably modified, reproduced the salient features of the normal human walking pattern, however there are a few significant differences in the control used to generate that walking pattern. As such, the model provided a useful framework from which to evaluate acceleration analysis based compensatory potentials – which are largely due to body orientation. The main significant difference in kinetics (control) compared to normal human walking data was the interchanging of hip and knee extensor function in early stance. The kinetic discrepancies signal areas of caution in follow up work that focus on evaluating model responses but do not affect the current work. Sensitivity analysis indicated that the model was quite robust – more so than what has been reported to date in literature. The simulation was able to withstand moderate to severe changes to groups of parameters while maintaining the basic walking pattern. Some of this benchmarking had been reported in the original paper[11] on which the current model is based however not in the same depth of detail as reported here. The sensitivity analysis had not to date been reported. Details of the benchmarking and sensitivity analyses follow.

1.2. Benchmarking

1.2.1. Rationale

The benchmarking analysis confirmed the extent to which the model reproduced the salient features of human walking as well as some of the underlying control mechanisms it employed. The subsequent acceleration analysis and control scheme assessments rely on the model having a

fundamentally similar walking pattern to that of normal humans. As our group was new to working with this, model it was necessary to carefully evaluate and document the model performance.

1.2.2. Methods

The methods for this benchmarking were simply to compare model values to published and in-house normal gait parameters. We evaluated temporospatial footfall parameters, muscle activation patterns (EMG), joint angles, joint moments and acceleration analysis profiles. Before presenting the model performance, a very brief overview of the model itself is provided. Also, some preliminary modifications made to the model – in order to make its performance more similar to normal human walking – are described.

1.2.2.1 Introduction to the model

A computer simulation of human walking is generally a set of mathematical equations compiled to represent the musculoskeletal system. In addition there is usually a separate sub-model that describes how the model interacts with the ground. Some models additionally have equations for muscle activation and contraction. Others have merely ideal torque motors at the joints. These equations are then "solved" using numerical integrators to calculate how the system moves as various muscles are activated. The present model had a hybrid muscle approach but that is not of critical interest. What is most interesting was the set of feedback loops in this model – that allow it to respond to external or internal perturbations. Briefly, there are neural oscillators at each joint that integrate sensory and motor information from the body to generate the proper control signals for walking. This rhythmic pattern is modulated by sensory

feedback and the higher brain center input to adapt and accommodate the control as necessary. It is noted this model was based on work carried out previously.[10] Details of the current model can be found both there as well as in a summary of the state of the art in computer simulation.[12] A graphical overview of the model is given in Figure 1 below. The subtleties of the inner workings of the control scheme are less central to the current efforts. What is more important is the final walking pattern that emerges from these inner workings.

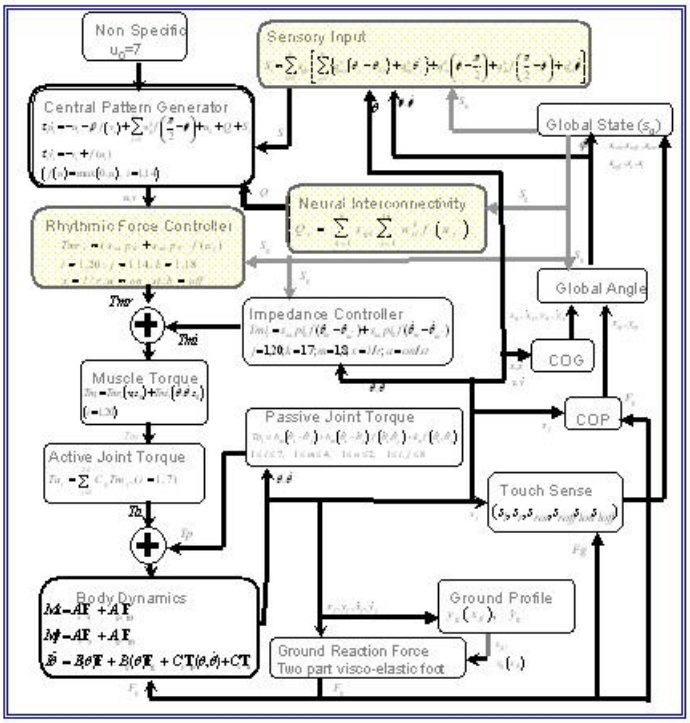
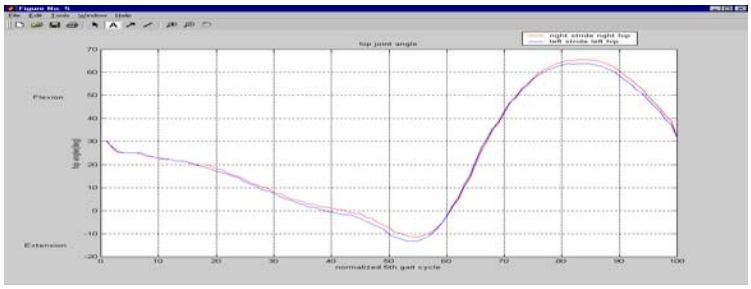


Figure 1. The basic structure of the model used in the current work. Adapted from Taga, G: Biological Cybernetics 73:97-111, 1995.

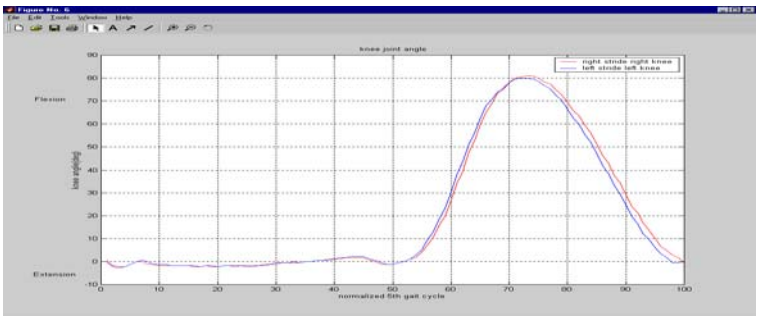
1.2.2.2. Initial model performance.

Our initial work with the model quickly revealed, upon casual inspection of the simulation data, that the model had a peculiar marching pattern rather than a smoother, level-ground walking pattern as shown below in Figure 2. As such, the model would be considerably less useful to evaluate normal gait and modifications to that pattern. This was addressed by

manually tuning specific model parameters as well as by mathematical optimization. In short, of the many parameters associated with the model (as can be seen in the model diagram of Figure 1), a few critical parameters were adjusted. This was done by a trial and error process. In the final analysis, the muscle strengths – as a group – as well as the tonic nonspecific input, u0, were adjusted. This resulted in a walking pattern as shown in Figure 3. This pattern was deemed a sufficiently adequate representation of human walking to allow subsequent work to proceed. It is noted that we also explored making customized models for each of the human subjects for whom data were collected. To do this, mathematical optimization techniques were explored and implemented. These methods require hundreds of millions of iterations to identify changes to combinations of model parameters. While the detailed methodology is less central to this project, the final results for one of the subjects are demonstrated below in Figure 4. In general, the subject-specific model generated a walking pattern that was nearly within a single standard deviation for joint angles of the stance phase leg over the entire stance phase. The overall error for the tracked angles was approximately 6.5 degrees RMS over the entire stance phase. These results compare favorably with the few other reports of subject specific computer simulations that are being developed.[13]

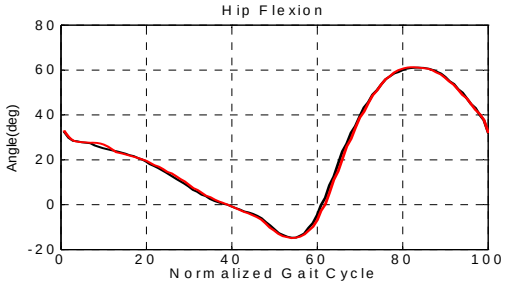


(a) Sagittal hip angle

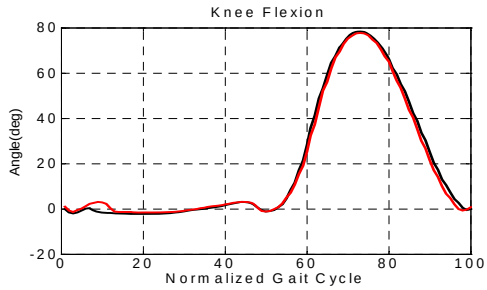


(b) Sagittal knee angle

Figure 2. Initial performance of model prior to manual tuning and optimization. Note the excessive hip and knee flexion in swing phases – indicative of a marching style gait.



(a) Sagittal hip angle



(b) Sagittal knee angle.

Figure 3. Modified performance of model subsequent to manual tuning. Hip hiking has reduced considerably from original (as published) model – indicating a much more normal gait pattern.

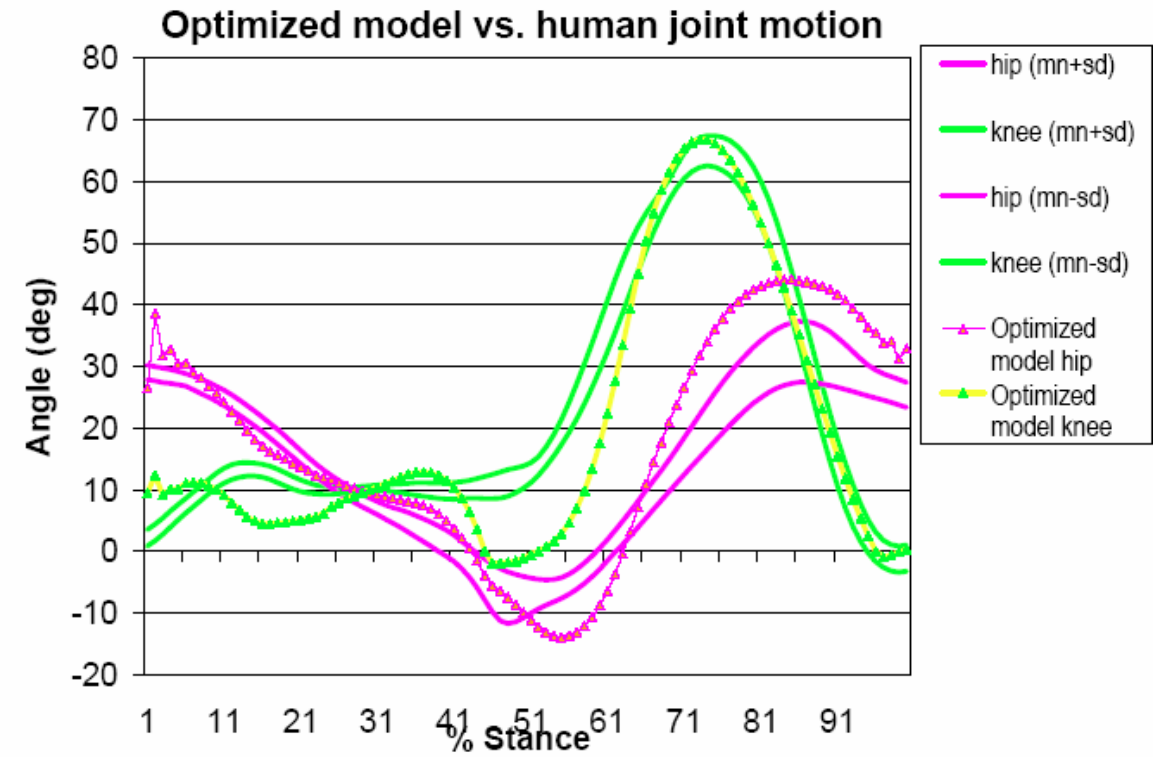


Figure 4. Optimized hip and knee sagittal angles. The reference data (solid lines) are from that of a single subject that was used as the goal in optimization of the model parameters. The excessive hip flexion noted in the baseline model has nearly been eliminated; the excessive knee flexion has been completely eliminated.

1.2.3. Results

It is noted that the modifications described in the last section were done to redefine the "baseline" performance of the model. All subsequent references to the baseline performance of the model refer to that of the model after the manual tuning was performed but do not include the subject-specific optimizations. Furthermore, it is noted that much of the benchmarking and sensitivity data were compiled for this project and reported in one of the graduate student thesis that resulted from the project. The graduate student and author of the thesis[14] are hereby acknowledged. The interested reader can find more details from that writing which is available through the Drexel University library. Some of the following text and figures are taken from that thesis.

A 70kg, 1.8m tall model was used for all analyses. Due to the nature of the model control scheme – a CPG that became "entrained" by the body and neural dynamics – there was a transient phase before steady state locomotion was achieved at around 10.5 seconds of the simulation. Data were analyzed for a complete gait cycle beginning at approximately 10.5 seconds. The temporospatial footfall variables are reported below in Table 5. A representative EMG comparison is shown in Figure 5. Joint angles and torques are shown in Figures 6 and 7. A representative acceleration analysis result is given in Figure 8.

Table 5. Temporospatial variables for the baseline model.

Variable	Model Value	Normative data
Cadence	92	78 to 144 steps/min [15]
Walking speed	1.02 m/s (0.57 statures/sec)	0.75m/s to 1.6m/s [16]
Stance/swing ratio	65%/35%	58-61%/ 42-39% [17]
Stride length	1.33m (0.74 statures)	0.25 to 1.5 statures [18]

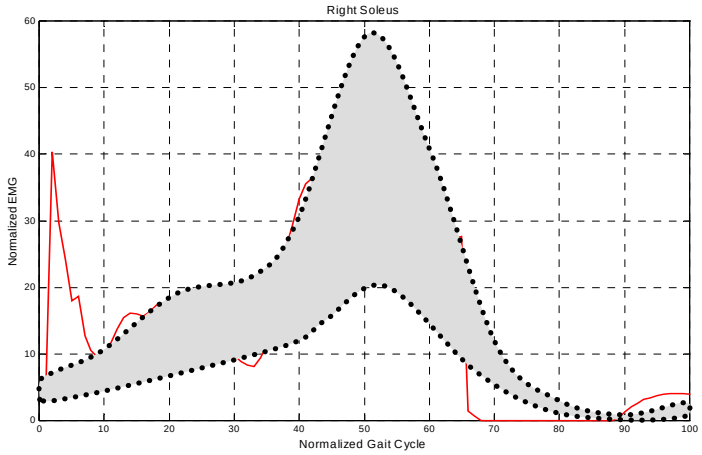
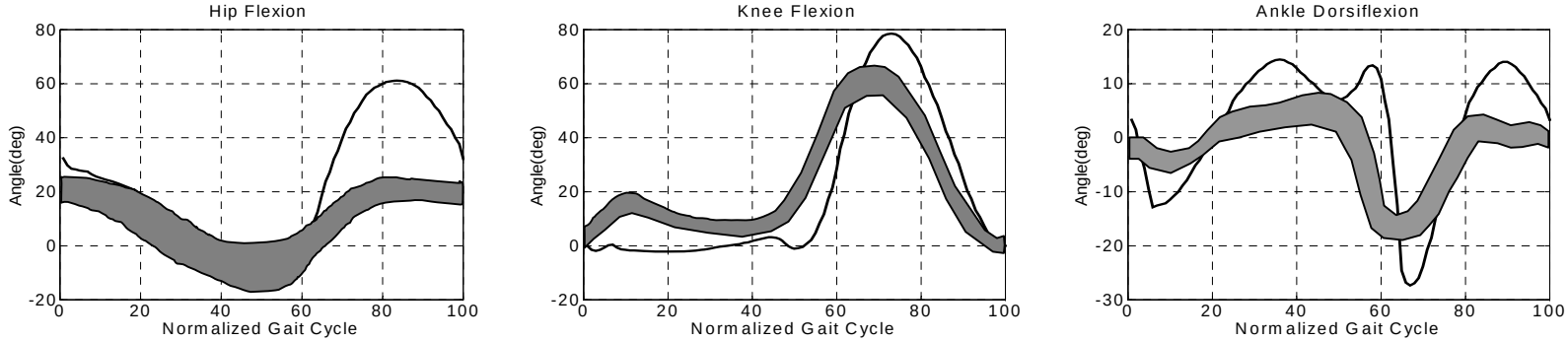
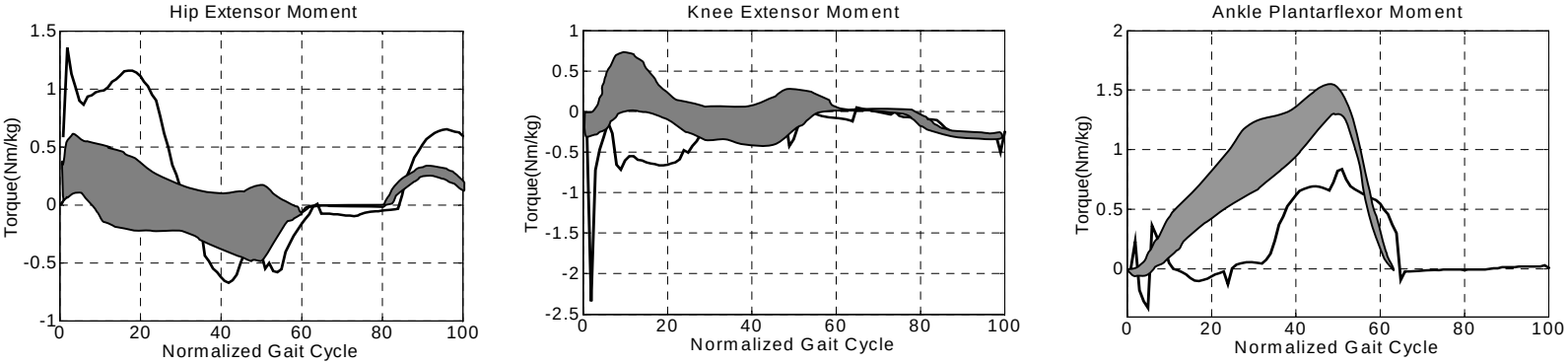


Figure 5. Representative comparison of model muscle activation to published normative data.



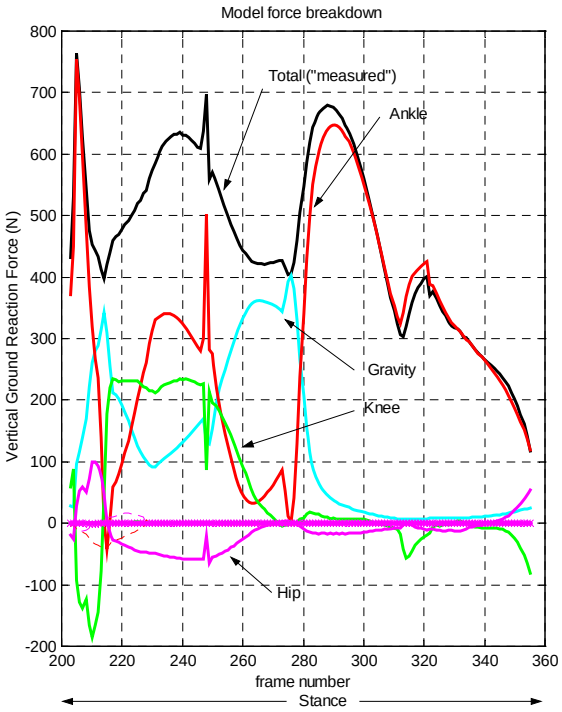
a) Baseline model hip flexion. b) Baseline model knee flexion. c) Baseline model ankle dorsiflexion.

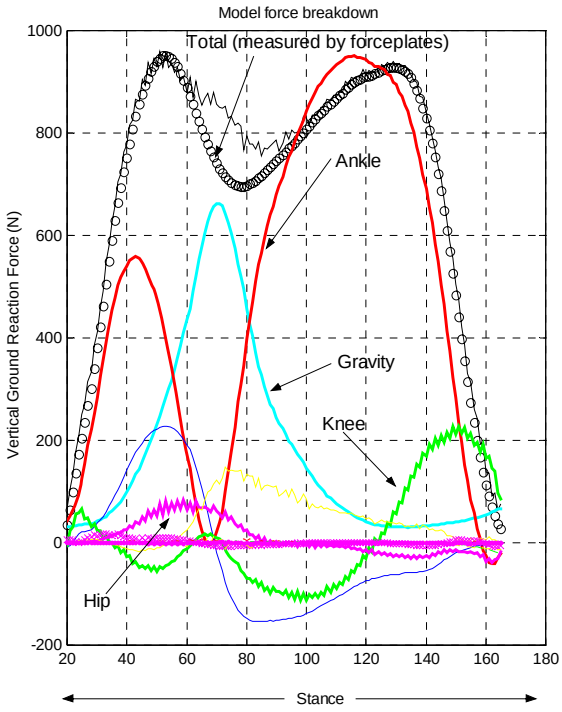
Figure 6. The hip, knee and ankle sagittal joint angles. Sign convention : Flexion >0.



a) Baseline model hip extensor moment. b) Baseline model knee extensor moment. c) Baseline model ankle planarflexor moment.

Figure 7. The hip, knee and ankle sagittal joint moments. Sign convention : Extensor moment >0.





1.2.4. Discussion

The majority of the benchmarking results were merely for assessment and documentation of model similarity to known parameters of human gait. Based on similarities, the model was deemed to adequately capture the salient features of gait. The most critical parameter for the intended current use of the model was the body orientations during stance phase. Once the model was updated via the manual tuning, the model tracked quite well the established "normal" walking sagittal joint angles. Interpretations of other parameters are done in the following paragraphs, but are not central to the subsequent objectives in this present work.

The model temporospatial parameters fell with the published ranges. There was a relatively wide range of values found for three of the parameters in normal literature. The model demonstrated a slightly longer stance duration. The muscle activation profile compared favorably to normative human EMG data that have been reported.[17] The activation of the right leg soleus muscle is shown to correspond well during loading response, late stance and preswing.

However it is seen a spike in soleus during initial contact occurs in the model but does not typically occur in human data. In general, this figure typifies the correlation – most important phases of activation are captured, the relative amplitudes also follow reasonably well, but sometimes an entire phase is abnormally missing or present. Additional graphical comparisons are provided in Appendix II. Due to arbitrariness in amplitude of muscle activation data, the published data were simply scaled to fit that output from the model – to emphasize and assist comparison of the activation timings.

The spike may be due to the abrupt change in model degrees of freedom when the leg goes from swing phase to stance. This is an impulsive event for which a large reaction force is generated at the ground – as is reflected in the model ground reaction force trace as well. In an effort to stabilize this impulsive force, the model generates a large muscle force near the disturbance – at the soleus muscle. The limited foot-floor model and muscle models likely contribute significantly to these types of uncorrelated responses in the model. The following major differences were noted in the model muscle activation profile: i) The gastrocnemius demonstrated the same spike at initial contact as soleus and otherwise did not show the moderate prolonged early activation as observed in normative data from approximately 0-20% of the stride, ii) tibialis anterior failed to show any activity during 90-100% of the cycle – when normally moderate activity is present, iii) vastus lateralis showed a prolonged high early activation phase from 30-55% of the cycle but did not capture the moderate activation from 90-100% of the cycle, iv) rectus femoris did not show the high early activation phase from 0-30% cycle or the moderate phase from 90-100%, v) biceps femoris did not demonstrate the high early activation from 0-50%.

The joint moment data show perhaps the most difference of the basic gait parameters normally evaluated in a motion analysis laboratory (Figure 3). While there tends to be considerable variability across even normal individuals in these quantities, the model data seem to be different even considering this. It is interesting to consider however that the summed joint moments for each leg may be relatively more similar than the individuals when comparing model to normative human data. The total support moment was thought to be a relatively fixed quantity.[19] As such, the model seems to be solving the support problem in a slightly different manner than the humans normally do. For example, the diminished extensor moment the knee in early stance seems to be made up for by the excessive extensor moment at the hip at the same time. This type of analysis is relatively subjective and speculative. The acceleration analysis evaluation also performed will further clarify differences in strategies used to generate and control the walking pattern observed.

The acceleration analysis sample result demonstrates that the fundamental means by which the model supports itself during walking is qualitatively and quantitatively similar to that observed in a human during stance phase – but it also highlights the key difference. Both panels (Figure 8) show a significant ankle contribution early and then a nearly total ankle contribution during mid-late stance. Both also show a large gravity contribution during midstance. These are the largest components and are common to both human and model. Both also show hip and knee contributions early, however it appears that the role of the net muscle action (moment) at each joint are opposite. The human shows a negative contribution from the knee and a positive one from the hip. The model shows the opposite. It is noted that there is no established "normative" value for human subject data at this point and so it is possible that the human subject data chosen are not representative of normal gait, but that seems unlikely. We have confirmed similar trends

in a very limited number of additional human subject data as well. It is also noted that the acceleration analysis models were slightly different for model and human subject data, and this could also be a small source of the observed discrepancies.

1.3. Sensitivity analysis

1.3.1. Summary

The sensitivity analysis was a means of assessing the limits of performance of the model. This clarified to which group of parameters and how much the model performance was dependent. Variables central to the four main functional groups in the model were assessed; the groups were neural interconnectivity, posture tone, sensory feedback, and muscle strengths. Establishing this operational range is important to further define and document the model and would be helpful in subsequent studies that may aim to assess the intrinsic model response to weakness. To assess the upper and lower limits of this range, all the variables in each group were sequentially incremented and decremented, respectively, until the model was no longer able to walk. The model exhibited operating ranges from 20% peak to peak (-10% to +10% over baseline values for rhythmic controller or active muscle strengths) to 150% (-50% to +100% for neural connectivity strengths). This suggested a quite robust control scheme for the current model and mathematical routines used to generate the simulation.

1.3.1. Rationale.

The purpose of this analysis was to assess robustness of the model to parameters variation. Though this is not critical to subsequent objectives in this study, it is an essential component of understanding a new model and documenting its abilities and limitations. It offers

a glimpse into how stable the model is – or how "precariously balanced" – in a mathematical sense – the model is. This characteristic will be much more important in follow up work to evaluate the intrinsic response capabilities of the current model to weakness.

1.3.2. Methods

Each variable in four key categories were changed as a group and the simulation was attempted to be run. If the model could produce a stable walking, the parameter group was further increased (or decreased) and the simulation reattempted until the model could no longer generate stable walking and fell over. The variable categories were rhythmic controller strength (pk), impedance controller strength (pik), sensory strength (qk) and neural interconnectivity strength (wk).

1.3.3. Results

The table below summarizes the operational range of the model with respect to the four major variable groups that were tested in the current sensitivity analysis.

Table 6. Results of sensitivity testing for model. Ranges below indicate changes over which model was able to maintain stable walking. Parameter group were changed one at a time.

Parameter group	**Stable range**
Strength of neural connection	*-50% to +100%*
Strength of sensory input	*-15% to +35%*
Impedance Parameters	*-20% to +50%*
Coefficient of rhythmic force controller	-10% to +10%

In addition to the mathematical sensitivity analyses, a perturbation test was done. The objective here was to evaluate the similarity of the response of the model to that of a human subject. A perturbation paradigm was chosen for which existing data already existed in our lab.

We elected to compare the model response to wearing a motion limiting brace on the ankle. A mathematical function was implemented to provide a restorative torque to the model's ankle when the ankle position extended beyond a set point in the normal range of motion. This is a common way that ankle foot orthotics are used in gait correction. The results of the model walking with and without the brace are shown below in Figure 9.

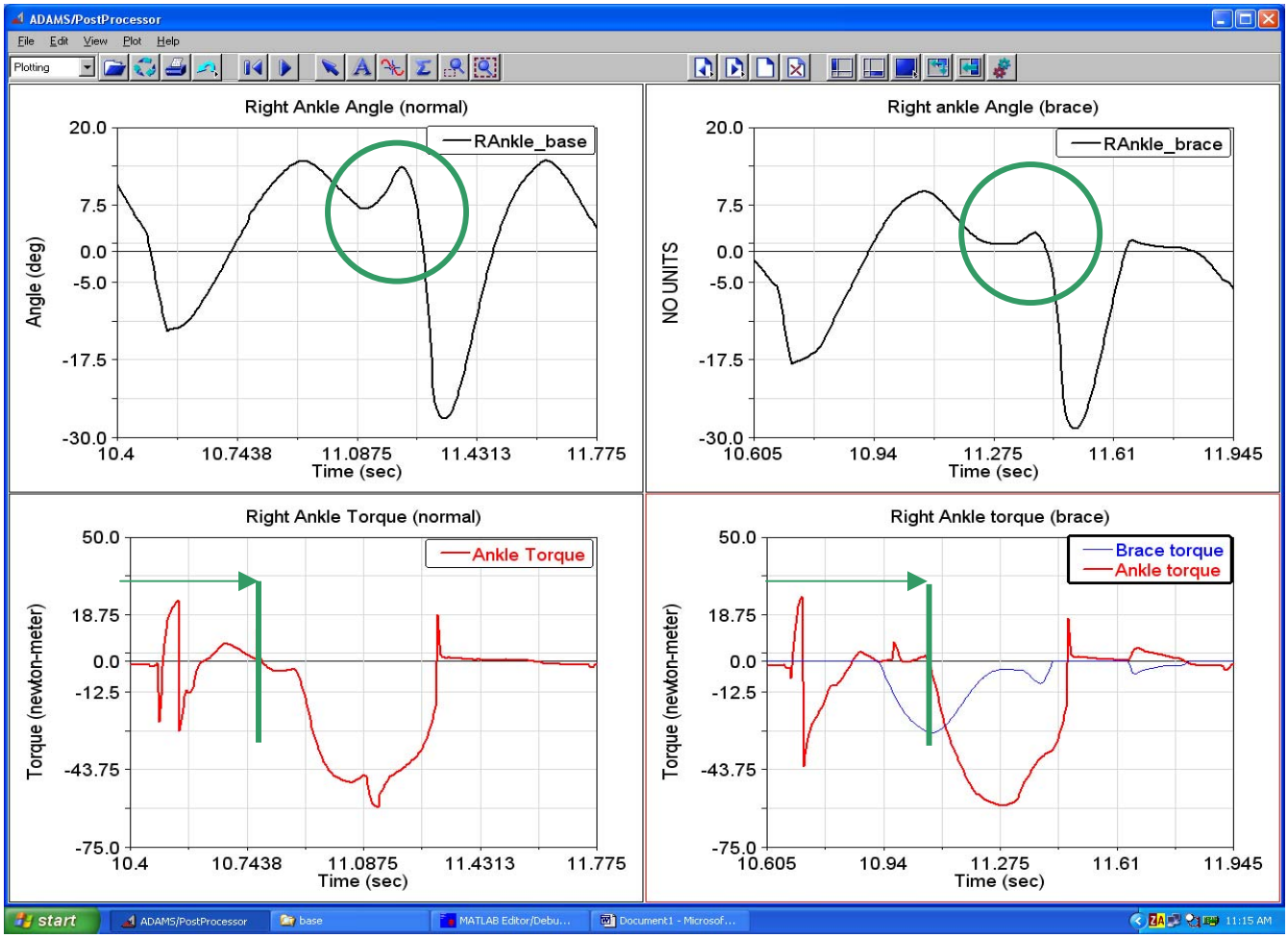


Figure 9. Ankle kinematics and kinetics of the model walking without (left) and with (right) an ankle foot brace that limited stance phase dorsiflexion. The top panel is the angle of the braced ankle [dorsiflexion >0], the bottom plot is the total joint torque acting at that ankle. In the braced condition (right), the ankle shows diminished midstance dorsiflexion – indicating the brace is set up correctly in the model. The additional blue line is the restorative or support moment provided by the brace. The model demonstrates an appropriate response by delaying generation of plantarflexor torque in midstance when the brace supplies that torque.

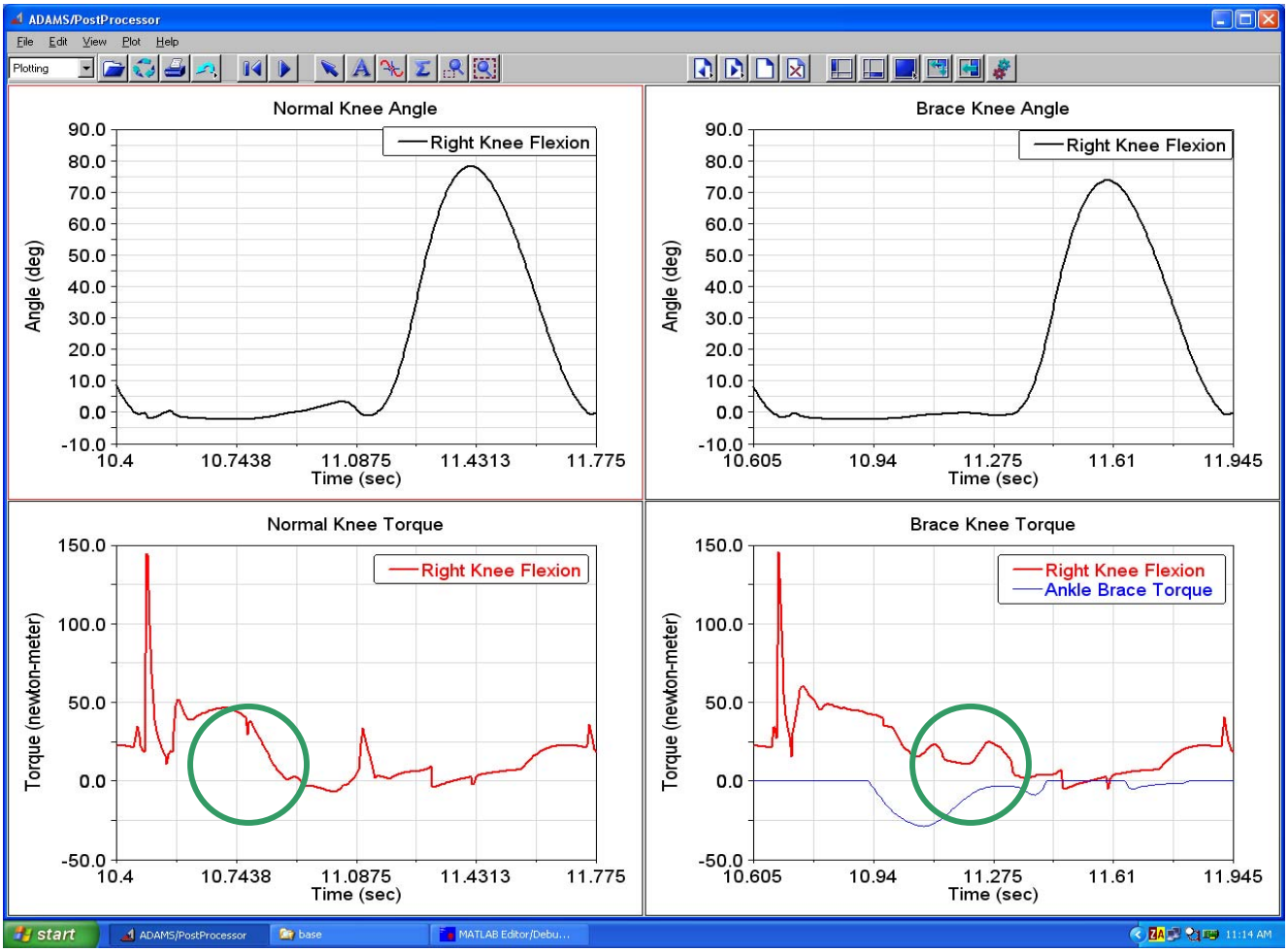


Figure 10. Knee kinematics and kinetics of the model walking without (left) and with (right) an ankle foot brace that limited stance phase dorsiflexion. The top panel is the knee angle of the braced leg [flexion >0], the bottom plot is the total joint torque acting at that braced leg knee. In the braced condition (right), the knee shows the model demonstrates again a feasible response by generating a flexor torque – to assist in flexion – during the time when the brace is providing a plantarflexor torque at the ankle.

1.3.4. Discussion

The model demonstrates robustness to a considerable amount of change in key variables. Perhaps the most interesting aspect of the model is its neural circuitry, followed by its sensory feedback components. Both of these subsystems demonstrated a sizable range of parameter space over which stable walking could be produced. This is important because the actual

coefficients used have little basis in any known physiological values – simply because the values have not been measured to date. It is reassuring that the model performance does not then depend too much on a very careful selection of these parameters – as their values are unknown. Again, this entire line of analysis will become critical for follow up work that aims to assess the intrinsic response of the model and its various control schemes to interventions and perturbations – such as weakness or braces. The intervention could similarly be strength or performance training as done in sports or military activity.

1.3.4.1. Perturbation testing – brace example

We further extended the model evaluation process by comparing response to a mechanical intervention. In short, the model was able to demonstrate feasible responses both at the local site of the intervention (ankle) as well as more remotely (at the knee). This is very promising for future work using this model or some variant of it – because the model has demonstrated not only robustness to perturbation but a similarity of response to the way humans behave. Further evaluating and refining this response can provide a tremendous tool for improving performance – whether in pathological gait, sporting or military tasks.

2. Interpreting the role of muscle groups during walking and the potential for compensation (Objectives 2 & 3)

2.1. Summary

In this portion of the project, we aimed to estimate the role of muscle groups to the specific movements they produce during walking. Such an objective cause-effect relationship has been missing, to date, from biomechanics – and is still only beginning to develop. Since this

work was proposed, a number of groups around the country have taken a similar approach – using acceleration analysis on real and simulated data – to further clarify the roles of muscles during walking. All of the groups consist of teams of researchers and are part of well established and highly regarded research programs. As such, our focus has expanded from simply generating these data on our own to compiling, reviewing and reconciling all available data with this common goal. While the model and assumptions used to calculate results surely affect the interpretation, there appears to be basis for development of loose consensus. A graphical tool to help in using these results is being developed.

2.2. Rationale

2.2.1. The development of an objective basis to identify primary deviations associated with weakness can clarify how best to select appropriate interventions to treat functional deficits observed in pathological gait. That was the original goal when proposing this portion of the work. Since that proposal, considerable additional research with a related focus has been published by others. The role of the present work has expanded to both 1) generating our own results as well as to 2) reviewing and reconciling our results with those of others. Through the comparison, we aim to identify which results are robust to the different modeling choices and analysis approaches and which results are sensitive or show differences based on them. In the course of our work and due in part to other recently published work, we have revised the methods slightly.

2.2.2. Knowing the potential of other muscles to assist in a particular function can help the clinician to identify where and how compensatory strategies may be implemented. This can

assist in determining the overall plan for correcting a dysfunctional movement pattern. It can also help minimize the chance that a beneficial compensatory strategy be unknowingly eliminated by a possible intervention. A wonderful example of this significance was given in a case analysis reported by researchers at the NIH.[20] This group showed how an individual with pathology had been using his opposite ankle to stabilize the knee. Another facility had prescribed the same patient an ankle foot brace previously to assist with a drop foot. However, the patient rejected the brace citing feeling occasional instability due to it. An acceleration analysis evaluate revealed the brace – which was intended only to affect the foot in swing - was disrupting the strategy this patient had developed to control the knee of the other leg. The clinicians prescribing this brace did not have access to the sophisticated biomechanical models to be able to identify the compensation strategy or the brace effect on it.

2.2.3. Historical note on changes to proposed methodologies.

Our original perspective aimed to explore how motions would change if a muscle was weakened and no compensation occurred. We proposed to use open loop simulations (OLS) to do this. Our preliminary results indicated that OLS are marginally stable at best and displayed a tendency to generate eccentric motions when controls were even slightly altered – as in implementing weakness. Aside from the fine balance of control signals required for OLS to recreate a movement, a larger limitation of this method was that the accelerations generated by each load change as body position changes. Thus using this method to evaluate "uncompensated" weakness was limited. Once the weakened muscle altered the body trajectory, the contribution from other muscles was altered despite the muscle forces themselves being unchanged. In addition, numerical sensitivity to changes in control lead to exaggerated

motions furthering this confound.

Induced position analysis (IPA) is a modification of the acceleration analysis methods. IPA clarifies how a muscle affects a motion by calculating its contribution to that motion through direct integration of IDA.[21] IPA is not subject to the limitations and constraints of OLS. Because IPA is an extension of AA, the contribution of each muscle group (net joint moment) is calculated independently of all others and the body positioning is reset to the actual for each time step. IPA is a useful paradigm to evaluate the role of a muscle to motions during walking without compensation. The process is an extra step in analysis beyond acceleration analysis alone. Our assessment of the IPA method suggests the relationship between calculated induced accelerations and its overall contribution to motions is straight forward. As such, there is no significant clarity obtained other than being able to quantify the actual contribution of a source in position/orientation degrees rather than in acceleration. The proportion of contributions of each muscle group (or more generally source) remained constant. Because of this, it was decided to interpret the acceleration analysis results directly to generate the functional deficits database.

Human subject data collected for the current study were analyzed. The computer simulation model showed a few key differences in the control profile compared to that of the human subjects tested in the current study. We compared acceleration analysis contributions of the model to human those calculated from subject data to determine this. This approach was thought to offer the best balance of accuracy, lowest computational cost and highest relevance and clearest meaning to understanding human muscle function.

2.3. Methods for evaluating muscle function

2.3.1. Literature review.

A number of highly relevant (similar) studies to the one proposed have been published since this project was initially conceived and accepted. For that reason, the methods have expanded to include to start, a review of those results. It is expected to critically evaluate and summarize both quality and the quantitative aspects of those results in the near future and update if necessary the findings of the current work.

2.3.2 Acceleration Analysis (AA)

The contribution of each muscle group to the acceleration of selected key motions of gait were objectively quantified. The key motions selected were stance phase knee and hip flexion and forward and vertical translation of the center of mass of the entire body**.** Standard acceleration techniques were implemented.[22] A 7 segment model consisting of bilateral feet, shanks, thighs and a combined pelvis-HAT segment was used for our analyses. The ankles were modeled as universal joints, the knees as revolutes, and the hips as spherical. Contact with the ground was modeled by a pin at the center of pressure during the entire stance phase.

2.3.3 A note on compensatory potential calculations

Fundamentally, this is still acceleration analysis as was described above however the main difference is that a unit value rather than the actual inverse dynamics calculated joint moment is input to the analysis. In our case, for a joint moment based analysis, the unit input was a 1Nm moment. This constant input value is applied for each joint moment – thus allowing us to evaluate the potential of equivalent muscle action at each joint to controlling the target

degree of freedom.

2.4. Results

The results of estimations of muscle roles during walking for the four degrees of freedom are presented in tabular form below. In Table 7, the model and key assumptions used by each group are also presented. For redundancy sake, this column was not repeated in subsequent tables. All analyses were reported for stance phase only – unless otherwise noted.

Table 7. Degree of freedom : Knee rotation

Group / Data source / Reference	Model	Findings
Present study	7 segment, lumped pelvis + HAT, universal ankle, revolute knee, spherical hip; pin at CP entire stance	Stance sagittal knee and frontal hip contributed to extension during entire stance with a very short assist (to 10%) from sagittal hip [a1f31]. Ankle and hip opposed early. By midstance, the ankle contributed to extension and gravity provided significant opposition [a1f31]. In late stance, ankle switched back to flexion, frontal hip fell off to zero [a1f31]. Brief period of frontal ankle contributing flexion as well (65-90%) [a1f31]. In other data, frontal ankle noted to provide significant extension assist through out much of stance [10-20%, 30-80%; a1f32]. **Potentials** [a1f avg]. Early stance : 25rad/s/s/Nm knee flexion per ankle df moment [10%], 11rad/s/s/Nm flexion per knee flexion moment, 10rad/s/s/Nm extension per hip ext moment (just after HS, ~2%) Midstance : Low (near zero) potential of ankle to control knee, 5rad/s/s/Nm extension per knee extension moment, 20rad/s/s/Nm extension per hip extension moment (~40%) Late stance : 2.5rad/s/s/Nm knee extension per pf moment [~70% (near peak Mpf)], 10rad/s/s/Nm extension per knee extension moment (~80%), 6rad/s/s/Nm extension per hip extension moment ** unable to assess accurately whether unintuitive effect of*

		ankle pf produced knee flexion early due to averaging process. Requires follow up analysis of individual trials.
Talaty 3 normal healthy Ss [23]	Single leg, planar, lumped pelvis, hat & swing leg; all revolute joints in body; pin joint at CP	Knee control varied considerably across stance phase. Ankle had considerable influence, sometimes greater than knee itself, to stabilize knee. Ankle potential was largest in early stance. Hip also had considerable though smaller and more constant potential to stabilize knee.
Kepple et al. 5 normal Ss [24]	7 segments, spherical ank/hips, revolute knee; fixed joint at CP during footflat, spherical when heel off	Calculated potential of ipsilateral moments only. The knee had the most potential control over ALL ipsilateral joints during footflat. All ipsilateral moments showed similar (order of magnitude) potentials at each joint. Noted that ankle PF could cause knee and hip flexion in late stance – but did not observe same phenomenon early as Talaty et al. did [23].
Siegel et al. 3 patho-logical Ss referenced to 1 Normal Ss, [20]	7 segment, spherical hips, revolute knees, universal ankles ; fixed foot during footflat, revolute at CP when heeloff	Evaluated subjects at single instant stance – approximately 15% (peak knee flexion for two Ss, estimate of "similar time" for two who didn't bend knees). Single normal subject found to generate 78% of knee extension acceleration from the knee extensor muscles and 21% from the hip extensors. Pathol subjects showed much different patterns of sources and relative contributions. All four did use hip extensors to generate knee extension acceleration, however magnitude of hip moment did not "predict" knee acceleration it produced.
Kimmel, et al. 20 control Ss [25]	7 segments w/ "gimbal joints" (spherical); HCtoFF and HOtoTO used "no translation" constraint; during FF, effectively welded foot	Used muscle actuated model. Calculated contribution of muscles to 3 leg joint rotation and whole body vert and fwd accels. All uniarticular muscles function "as expected" but some biarticular function is counterintuitive. Specifically, rectus femoris did expected extension at knee but did extension not flexion at hip. Long head biceps femoris did expected extension at hip but extension not flexion at knee. Gastroc was only biarticulate (of the three) that did "expected" at both joints. All uniarticulars did as expected. They were vastus intermedius, iliacus, soleus, biceps femoris short head.

Neptune et al. [26, 27]	Sagittal model.	Assessed vertical and forward component of reaction force at hip joint to calculate power (P = **F** dot **v**) of simulated (not actual human subject) data. Uniarticulate knee extensor (vastus intermedius) accelerates knee into extension. Rectus sign if contrib. to extension (in late stance only?)
[28]		Gmax, Vastii produce extension in early-mid stance,sol in mid-late stance. Relative magnitudes differed from Arnold work (latter speculated due to diffs in musculoskeletal model).
[29]	3d simulation (not real data). Anderson model: 10 seg, 23 dof, 3d, 54 muscles. Lumbar, hips spherical, knee revolute, ankle (subtalar) universal, metatarsal hinge; 5 element VE foot	Single limb stance. GMAX, Soleus, Vastii produce extension. Adductor magnus also had potential to produce extension. GMAX > Vastii (per unit force) – interesting that can do more from hip. Iliopsoas and adductors (Sartorius, TFL, adductor brevis, adductor longus and pectineus) and gracilis can produce hip flexion (mid and late stance). Note that biarticulars (hams, rectus and gastroc) had smaller potentials to control knee – due to fact that both "ends" of muscle produced opposing effects at knee – so overall effect was diminished.

Notes: CP = center of pressure.

Table 4. Degree of freedom: hip rotation (extension)

Group / Model / Data source / Reference	Findings
Present study	Ankle contributes to flexion mostly throughout stance – with exception of 60-75% [a1f avgs]. Knee contributes flexion early (0-10%) but then mostly extension for remainder of stance [a1f avgs]. Hip contributes extension early (to ~10%) but then flexion for remainder of stance [a1f avgs]. **Potentials [a1f avg]**. Early stance : 8rad/s/s/Nm flexion per ank df moment [10%], 5rad/s/s/Nm

	ext per knee ext moment [10%], 5rad/s/s/Nm ext per hip ext [just after HS, ~2%] Midstance : *3rad/s/s/Nm flexion per ank df moment [40%]*, 2.5rad/s/s/Nm ext per knee ext moment [40%], 12rad/s/s/Nm flex per hip flex moment [40%] Late stance : ~1rad/s/s hip ext per ank pf moment [80%], 5.5rad/s/s/Nm ext per knee ext moment [80%], 3.5 rad/s/s/Nm flex per hip flexion moment
[25]	Uniarticulate knee extensor (vastus intermedius) produced knee extension accel [26, 30]
[26, 27]	Uniarticulate knee extensor (vastus intermedius) accelerates hip into extension. Rectus signif contrib. to extension (in late stance only?)
[29]	Single limb stance. GMAX, Soleus, Vastii produce extension. GMAX > Vastii (per unit force). Iliopsoas and adductors can produce flexion. Hamstrings (producing knee flexor and hip extensor moment) accel hip into extension (but ~no effect on knee). Adductor magnus also can produce extension. Sartorius, iliopsoas, TFL, adductor brevis, adductor longus and pectineus and gracilis also can produce hip flexion (mid and late stance).

Table 9. Degree of freedom : whole body vertical (support)

Group / Model / Data source / Reference	Findings
[25]	Uniarticular hip flexor (iliacus) produced collapsing acceleration Uniarticulate knee extensor (vastus intermedius) produced support Biarticular knee extensor (rectus) produced support.
[26, 27]	Used contribution to trunk accels for support. GMAX main contributor to support early. Uniarticulate knee extensor (vastus intermedius) provided support in early stance but tailed off to no effect by mid stance. Biarticular knee extensor (rectus) produced support but relatively negligible compared to other muscles they studied. Uniarticulate ankle PF (soleus) and biarticulate (gastroc) provide support through out single leg stance.
[30]	Uniarticulate knee extensor (vastus intermedius) contributed to support after foot-flat and in varying amounts through remainder of stance. Biarticular knee extensor (rectus) produced collapse upto ~10% stance and then support remainder of stance.

Table 10. Degree of freedom : whole body forward (propulsion)

Group / Model / Data source / Reference	Findings
[25]	Uniarticular hip flexor (iliacus) produced braking acceleration Uniarticulate knee extensor (vastus intermedius) produced braking Biarticular knee extensor (rectus) produced braking.
[26, 27]	GMAX main contributor to propulsion early. Uniarticulate knee extensor (vastus intermedius) produced braking for early stance – tailed off to no effect by mid and late stance. Biarticular knee extensor (rectus) produced braking but relatively negligible compared to other muscles they studied. Uniarticulate ankle PF (soleus) and biarticulate (gastroc) provide braking in early single leg stance. In midstance, SOL accels trunk, GAS brakes it. In late stance, both accel trk forward. Rectus femoris makes signific contrib. to propulsion in late stance.
Kepple et al. [22]	Ankle main contributor to forward propulsion during late stance. Estimated forward as acceleration of trunk.

2.5. Graphical presentation of key effects during snapshots of the stance phase.

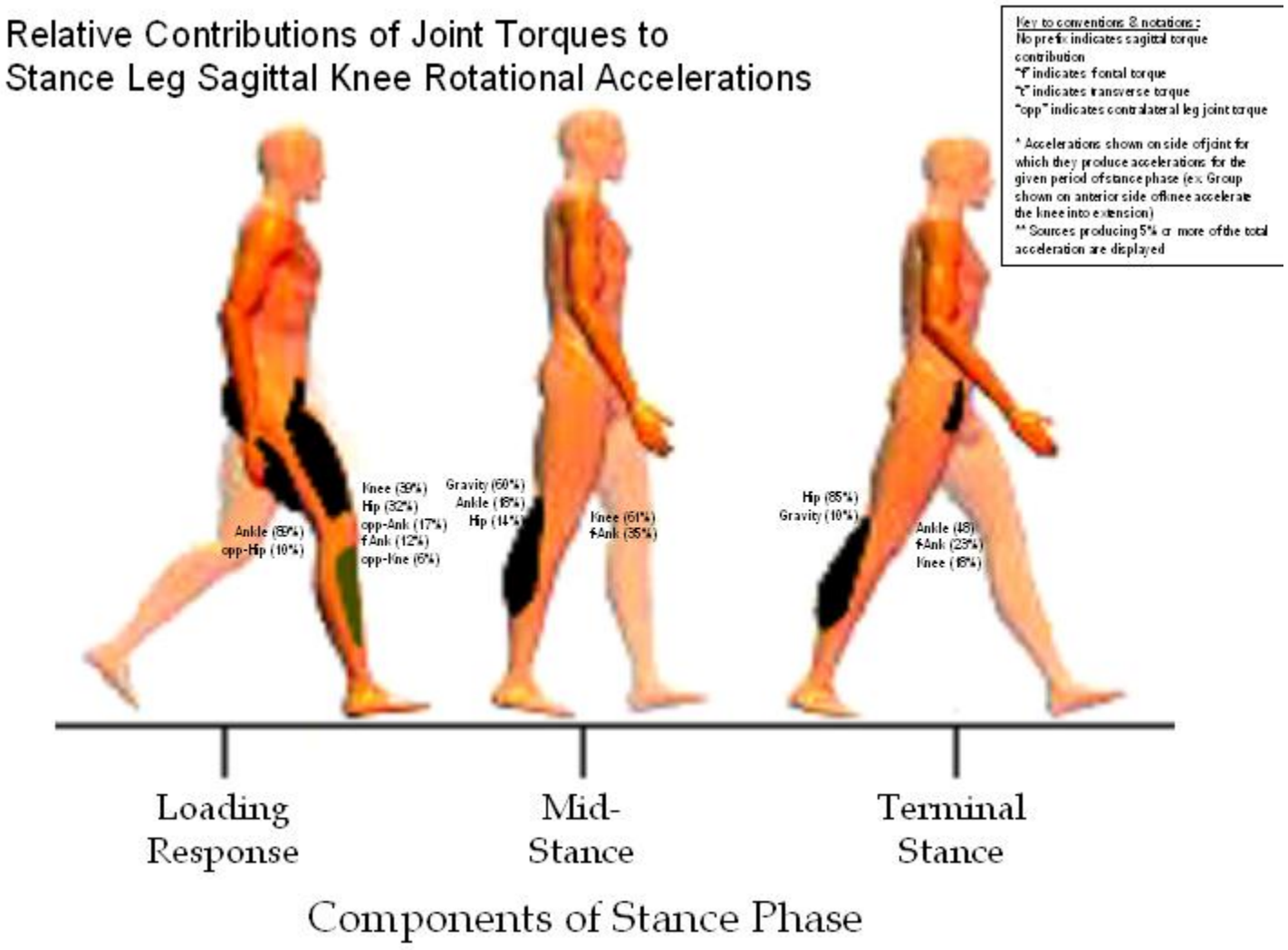


Figure 11. Graphical database of the major relative contributions of joint torques and other sources during three distinct periods of the gait cycle.

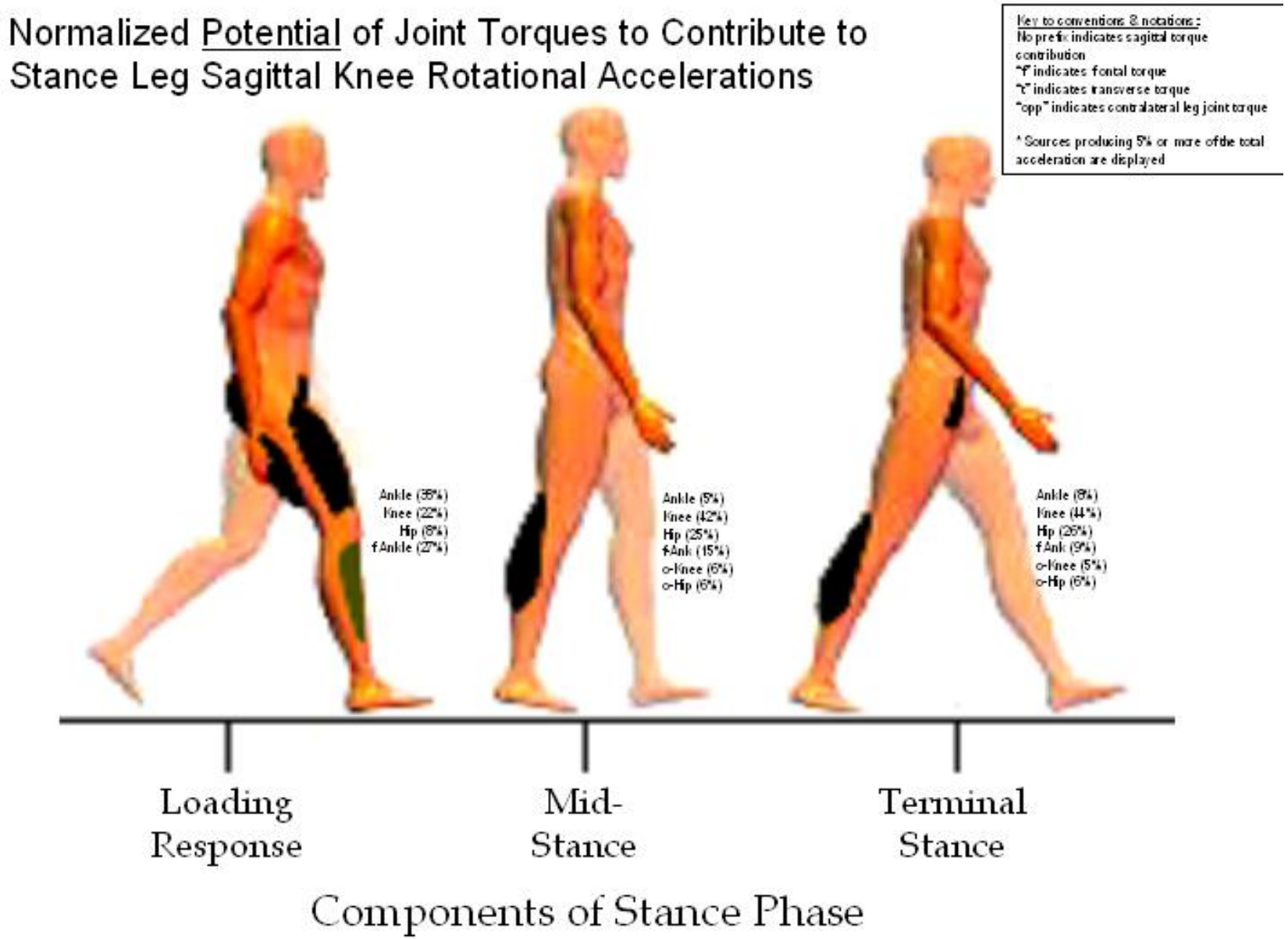


Figure 12. Graphical database of the major potentials for joint moments to contribute to sagittal joint rotation during three distinct periods of the gait cycle.

Note on Compensatory Potentials

2.1.1. Compensation potential data demonstrate how other muscles may be used to assist weak or otherwise dysfunctional ones. It is worth repeating that the overall process by which compensatory actions is complex and not at present clear. For example, the gluteus maximus may demonstrate the highest potential to assist weak knee extensors but may have undesired effects on forward progression making them an unacceptable choice. Or perhaps, yet another muscle (group) may be required to offset the braking effects of the gluteus. How the muscle combinations are chosen is largely unknown to us at present. Furthermore, this framework of interpreting the muscle potentials is necessarily developed on the premise of a given body orientation. Muscle potentials change as body orientation changes. Patients routinely employ compensatory strategies that involve altering – sometimes grossly – body orientation in pathological gait. In these cases, a new set of potentials would exist.

3. Implementation of Mechanical-based compensation scheme (Objective 4)

3.1. Rationale

3.1.1. To estimate what fraction of a selected compensatory strategy could be explained by calculated contributory potentials. This was assessed in healthy normal individuals in response to a short-term focal weakness. A casual observation in previous work (unpublished laboratory results) suggested that polio survivors generated responses loosely in line with their mechanical potential to do so. This objective aimed to assess whether that was true by evaluating this response in a more quantitative manner. The

ultimate goal is to clarify exactly how a compensatory response may be generated. What are the priorities in this response? Knowing this can help predict how patients may respond to certain interventions and what repercussions (such as overuse, joint damage, etc.) may result from selected strategies.

3.2. Methods

3.2.1. We posed the problem an over-determined system of algebraic equations where the independent variables were joint torques and the dependent variables were joint and body segment accelerations. We used mathematical techniques to solve this system – effectively calculating how all the joint torques, except the one designated as "weak", would change in an attempt to maintain the original system accelerations. In essence, we are operating on the assumption that subjects would try to maintain their baseline walking profile after we introduced temporary weakness to one muscle group. We know from clinical practice that this is often not the case, as patients are not able to maintain "normal" walking once they have obtained functional deficits. However, the above assumption may well hold for healthy normal subjects with a short term focal weakness – and is an ideal place to begin our exploration of compensatory response.

3.2.2. Detailed methods are described in the code used to evaluate the human subject data. The code is provided in Appendix III. [pi_compCp2.m]. In short, the system of accelerations was compiled. Using acceleration analysis decomposition from the previous objective, the component of acceleration due to the muscle group that was weakened linearly according to the amount of weakness generated. This left a deficit in acceleration. The

acceleration analysis potentials, from objective 3, were essentially the transfer function between the joint torques and their ability to produce acceleration. Thus, changes in joint torques to make up the acceleration deficit could be estimated by solving this over-determined system.

3.3. Results

3.3.1. Solution of system of algebraic equations

3.3.1.1. Accelerations reconstructions

An important prerequisite to evaluating SAE solutions – particularly in an over-determined system – is how closely the desired outcome was reached. In this case, our outcome was reconstruction of the original acceleration profiles. When a new control pattern is obtained (modified joint torque patterns), they are multiplied back into the inertia matrix to determine what acceleration results. Figure 13 below shows the original (old) and two solution profiles. The "new" profile matches the desired quite well.

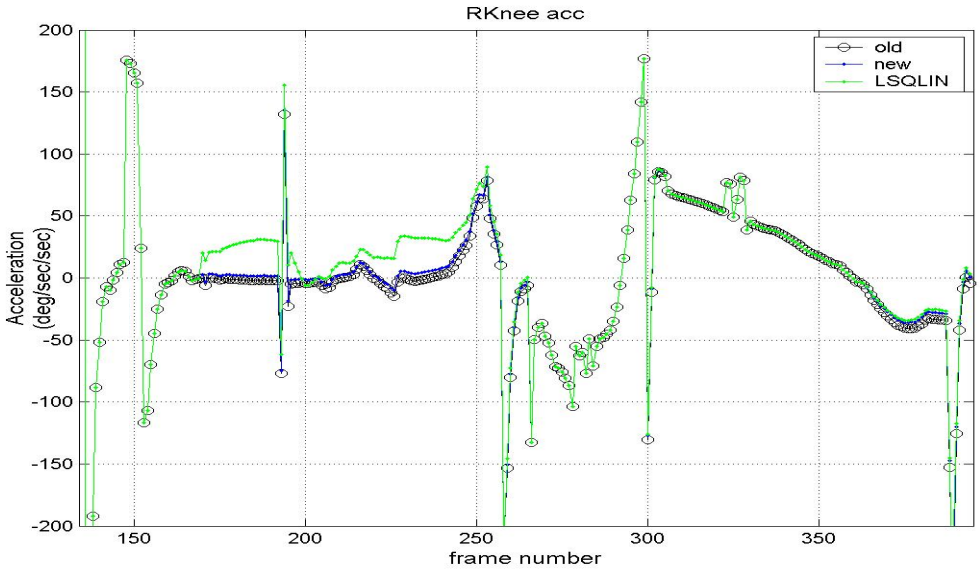


Figure 13. Reconstruction of the desired (baseline) acceleration profile using two solution methods outlined in Appendix III.

3.3.1.2. Calculated joint moments

The main outcome of this analysis was the joint torques. They represent the change in control strategy used to compensate for the weakness introduced. Figure 14 shows a sample of the calculated joint torque profile. It is seen that the torque has changed significantly from its baseline value – indicating this particular torque played a major role in the compensation process. In this particular case, the weakness was introduced at the ankle, so it is expected that the knee – the most proximal joint – would be significantly involved to assist that weakness.

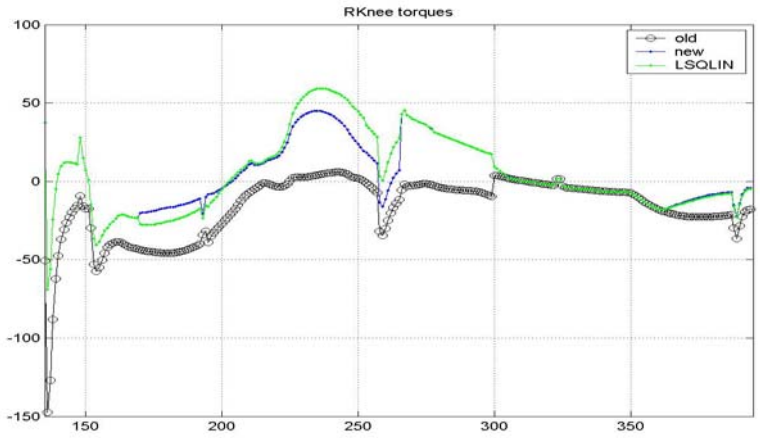


Figure 14. Estimated changes in the knee torque to support weakness in ankle plantarflexors.

3.3.2. Comparison of calculated response to human response for a single human subject

3.3.2.1. Body forward acceleration in late stance

The results indicate that the potential to contribute or make up for the deficit introduced by the weakness only accounted for a small fraction of the observed response. The source with

the largest potential showed only a modest contribution to recovering acceleration lost due to weakness. The 2nd and 3rd largest components showed the largest compensations.

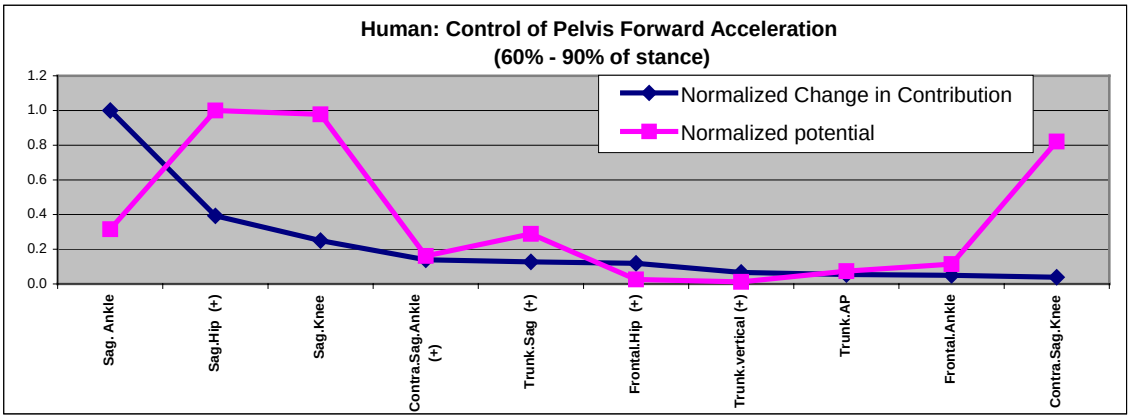


Figure 15. The major contributors to pelvis forward acceleration along with the potential of the contributors, during late stance for a single subject.

3.3.2.2.Hip sagittal rotation in late stance

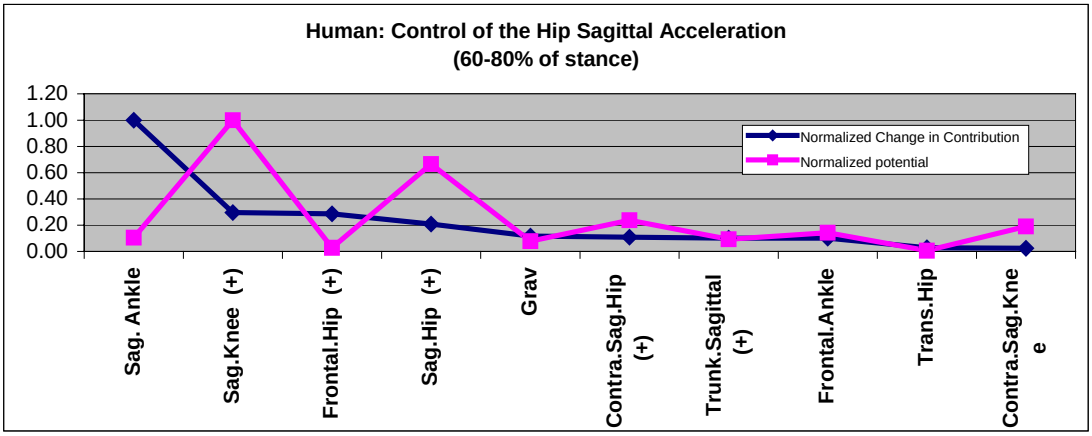


Figure 16. The major contributors to hip rotational acceleration along with the potential of the contributors, during late stance for a single subject.

3.4. Discussion

Determining the role of mechanics in compensatory strategy selection can help to establish an objective basis for prediction of response of a patient to an intervention. The clinician's selection of corrective measures in pathological movement is often a trial and error process. One significant contributor to this variability is lack of sufficient predictive models and tools to assess potential response to a possible intervention. It is not expected that the proposed scheme will account entirely for the observed compensatory response. However, it will surely account for some fraction of the total response. Knowing this can help us to predict responses and to focus our search for the remainder of the compensatory process. Furthermore, it may be that certain patterns of invariants in the compensatory response can be identified through analysis begun here.

The current analysis is in early stages. Only the model and a single subject have been evaluated and in a limited capacity. The methodology seems to be implemented adequately. The reconstructions are reasonably close (within 5% for most degrees of freedom and for most times) suggesting validity of the obtained solution. The calculated joint torques are piecewise continuous (i.e. physiological) and of reasonable magnitudes. They show moderate changes as are seen by real patients employing compensatory strategies. The early data suggest the mechanical potential of a joint to assist a focal weakness only account for a small percentage of the observed response. It is however a bit premature to quantify this amount as the analysis has only been run for a single subject. Data analysis continues.

It is worth noting limitations at this point – to put the results in proper context. The current methods can not account for how strategies that involve changing performance kinematics may occur. In its best perspective, the current tools can only account for a fraction of

the response for shifts in control strategy that aims to restore a known (baseline) performance kinematic profile. And it has only been evaluated in healthy normal subjects for a short term focal weakness. This represents a significant amount of restriction on applicability of the present results to the desired goals. Nonetheless, the results represent a step toward the development of tools to establish a much needed objective framework.

KEY RESEARCH ACCOMPLISHMENTS:

1. The results of this study confirmed the link between lower extremity weakness and different measures of fatigue among polio survivors and older controls. Body weight was also determined to be an important factor for controls and polio survivors with PPS. However, this relationship did not prove to be generalizable to polio survivors without PPS. Differences in the group size may have been a confounding factor.
2. Through this research, we were able to determine that fatigue in polio survivors and controls is closely related to activity level. Steps at high and medium levels were significantly associated with fatigue level in all three groups. In general, polio survivors with PPS walk less However, their maximum walking capacity is lower and the difference between their normal performance level and their maximum performance level is smaller. Therefore, they are at higher risk for symptoms commonly associated with overuse, like pain and fatigue.
3 Pain severity in polio survivors without PPS was associated with season and activity level. Severity levels were lower in the winter when you would expect people to be less active and increased with the number of high steps. However, season was not a significant factor in the group models for daily step activity for either of the post-polio groups. It is possible that

knee extensor strength in the weaker leg was the limiting factor. However, further research is needed to confirm this hypothesis.

4. The benchmarking and sensitivity of a forward dynamic simulation model of bipedal walking was accomplished. Extensive additional documentation is available if desired.
5. A graphical chart to describe the role of muscle groups during walking (referred to as a functional deficits database in the original proposal) was produced and is reported for the sagittal rotation of the knee in the results section. Other motions, including the sagittal hip rotation, and body forward and vertical translations are forthcoming. It is expected that these results will be publishable either in a clinical or a research-clinical journal – when additional subjects have been added to make the results more robust and general.
6. Assessment of compensatory potentials of muscle groups to motions during walking was reported. In addition, this was presented in the same graphical template used for the above objective, for easier interpretation and reference. It is expected that these results will be publishable either in a clinical or a research-clinical journal – when additional subjects have been added to make the results more robust and general.
7. Human subject data were obtained through a protocol that involved administering a lidocaine block to generate acute focal weakness. All subjects were consented and no adverse events of any kind were observed. Strength and gait analysis data were obtained for eight total subjects. Additional subjects were interested to participate but time and resources did not allow further testing still.
8. The routines to calculate the mechanical compensatory scheme were coded into Matlab programming environment. Routines were tested on the data of two of the test subjects.

The results allow assessment of the mechanical component of the observed compensatory response. It was originally stated that a comparison between the predicted and actually observed data would be made and the data scored for similarity. This aspect of the study has not yet been completed. It is expected that this analysis will continue until the stated objective has been completed.

REPORTABLE OUTCOMES:

Presentations:

- Steele W, Talaty M, Esquenazi A. Acceleration Analysis to Quantify Compensation in Amputee Gait: The Effect of Prosthetic Foot Position in a Trans-tibial Amputee. Abstract submitted for presentation at the 33rd Annual AAOP conference to be held in San Francisco in March 2007.

- Patel M, Talaty M, Coulter T, Esquenazi A. Klein M. Evaluation of the compensatory response of a CPG-Based Model to ankle plantar flexor weakness. Poster presented at the Biannual International Society of Biomechanics Conference held in Cleveland, OH, August 2005.

- Klein M, Costello R. Analysis of Activity Patterns and Health Status in Polio Survivors and Older Adults with No History of Polio: Preliminary Results. MossRehab Research-in-Progress Colloquium, MossRehab Hospital, Philadelphia, PA in December 2004.

- Patel M, Talaty M, Klein M, et al. Evaluation of a computer model to simulate human walking and understand compensatory behavior for muscle weakness. Research Recognition Day, Albert Einstein Medical Center, Philadelphia, PA, May 2004.

- Talaty M. Models for Gait Analysis. 5th SIAMOC (Societa Italiana Di Analisi Del Movimento in Clinica) Congress, Loano, Italy November 2004.

- Talaty M, Patel M, Coulter T, et al. Physics-based computer simulation to assist in the understanding of mobility dysfunction and rehabilitation. Research in Progress Symposium, MossRehab Hospital, Philadelphia, PA, September 2003.

- Talaty M, Patel M, Esquenazi A, Klein M. The effect of variation of neuromusculoskeletal model control parameters on performance during simulated human walking. IX International Symposium on Computer Simulation in Biomechanics, Sydney, Australia, June 2003.

- Patel M, Coulter T, Talaty M, Esquenazi A, Klein M. Development of a computer model to simulate the effect of localized muscle weakness on walking. Gait and Clinical Movement Analysis Society Eighth Annual Meeting, Wilmington, Delaware, May 2003.
- Patel, M, Talaty, M, et al. "Evaluation of a Computer Model to Simulate Human Walking and Understand Compensatory Behavior for Muscle Weakness," Albert Einstein Healthcare Network, Annual Research Symposium. April 2003.

- Talaty, M. Computer Modeling and Simulation in Gait Analysis. PM&R: State of the Art Reviews. 16(2): 339-360, 2002

Education/Training Outcomes:

- Graduate students funded in part by this grant

  - Mausam Patel. Completed MS thesis entitled, "Evaluation of the Compensatory Response of a CPG-based Neuromuscular Skeletal Model to Localized Muscle Weakness" in June 2004.

  - Thomas Coulter. Currently deployed as an activated Reservist in Kuwait (2nd activation during this project). Expected to return in 6-12 months to complete thesis work.

  - Wilson Steele. Completed MS work entitled, "Development and Validation of a Three Dimensional, Eight Segment Mechanical Model to Determine Contributions of Individual Loads to Acceleration In Human Sit to Stand Motion" in June 2006. Part of his analytical development led to improvements in the analysis codes used in the current project.

Manuscripts in Progress:

- A manuscript entitled, “Response of a generic CPG based model to localized muscle weakness”.
- A manuscript entitled, “How able bodied subjects compensate for acute focal lower extremity weakness during walking”.
- A manuscript entitled, “The impact of adding thorax motion to the interpretation of the role of the joint moments during normal walking”.
- A manuscript entitled “Factors associated with community walking activity in polio survivors and older controls”.

CONCLUSIONS:

1. The results of this study indicate that polio survivors are less active in terms of walking activity than older controls. The amount of walking activity among polio survivors is not affected by season, age or body mass index as in controls. As expected, strength in the weakest knee extensor was an important factor for all three groups.

2. One larger focus is in the development of a computer simulation that embodies a physiologically inspired control scheme and associated circuits (loops) that responds in a similar manner to a human. Such a tool would allow for many questions on performance, restoration of functional movement, optimization of intervention, etc. to be explored with a minimum time and cost investment. The model used in the current work has demonstrated promise in this area. It has neurologically inspired control circuitry, walks in a manner that

is fundamentally similar to that of a human, is stable and robust to considerable perturbation, and has shown signs of responding in a manner similar to that observed in actual human data. To what extent these similarities hold, what factors circumscribe the limits of applicability of this model, and exactly which control circuits are responsible for what specific aspects of the model response are some of the key questions that remain to be answered.

3. Combining elements of the neurologically inspired control circuits present in our model into other computer models, for example the SANTOS virtual soldier being developed at the University of Iowa, would very likely improve the fidelity of predictions being made by that model.

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APPENDIX I

Project Staff Members

Mary Klein, PhD Principal Investigator

Alberto Esquenazi, MD	Co-Investigator
MaryAnn Keenan, MD	Co-Investigator
Mukul Talaty, PhD	Research Engineer
Roberta Costello, MSN, RN	Project Manager
Jennifer Kuehl, PT	Physical Therapist
Janet Wechsler, PT	Physical Therapist
Mausam Patel	Research Assistant
Wilson Steele	Research Assistant
Thomas Coulter	Research Assistant
Ann Louise Simone	Research Assistant
Gemma Baldon	Database Designer
Len Braitman, PhD	Biostatistician

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APPENDIX II. The activity of the major muscle groups of the model as compared to published normative data.

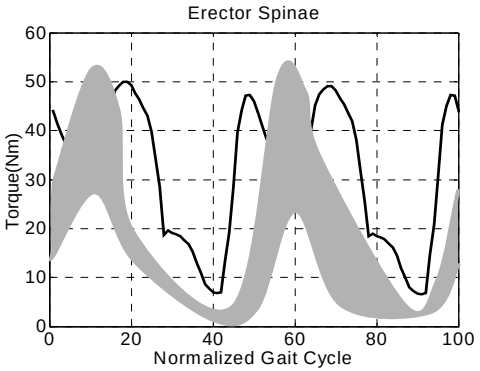

Figure 1a. Baseline model erector spinae activity

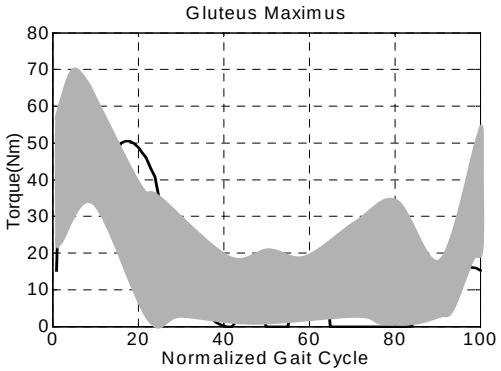

Figure 1b. Baseline model gluteus maximus activity.

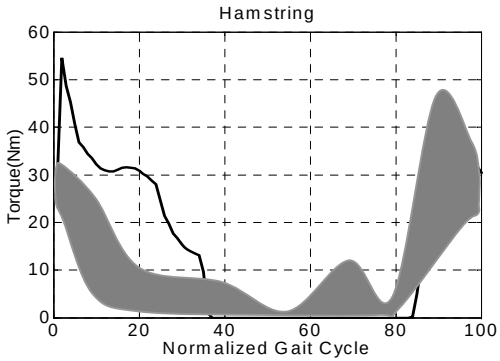

Figure 1c. Baseline model hamstring activity.

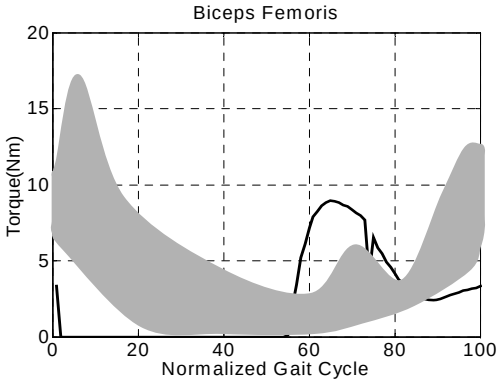

Figure 1d. Baseline model biceps femoris activity

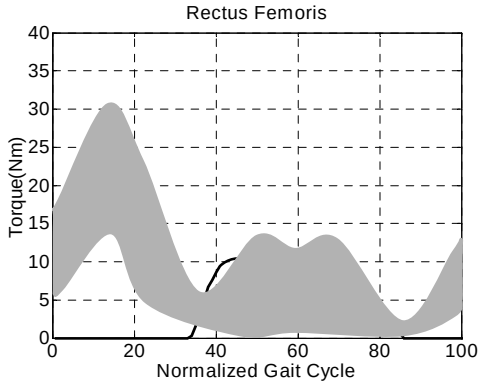

Figure 1e. Baseline model rectus femoris activity.

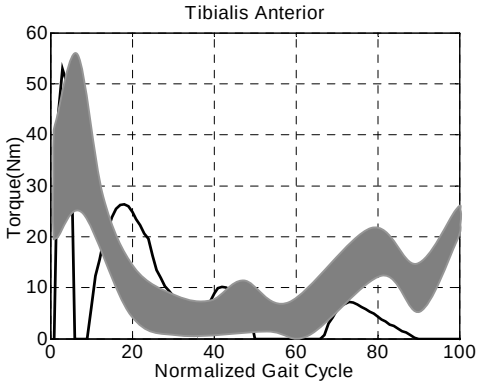

Figure 1f. Baseline model tibialis anterior activity.

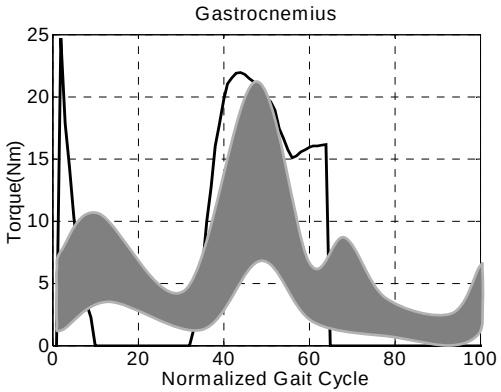

Figure 1g. Baseline model gastrocnemius activity.

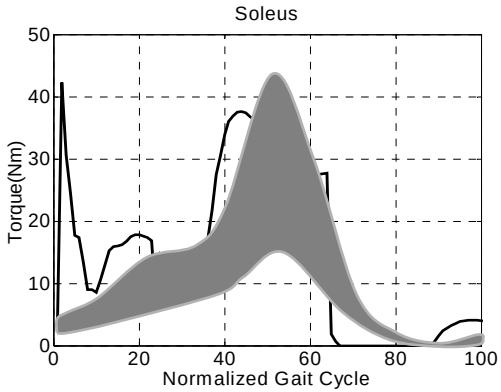

Figure 1h. Baseline model soleus activity.

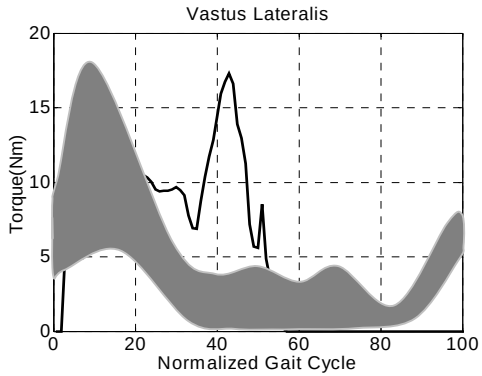

Figure 1i. Baseline model vastus lateralis activity.

Appendix III. Matlab codes used to calculate mechanical aspect of compensation response

```
% Program PI_cmpCp2.m
%
% A code to calculate the compensation scheme based on the hypotheses that the
% body orientation, position and accelerations of the compensated walk would be
% the same as the non-weakened walk. The IDA results were used to calculate the
% compensation scheme.
%
% T. Coulter, M. Talaty
% 07/13/2004, revs 8/21/2006


clear all;
clear all;
filename=dir('IDAresults_taga*.mat');
filename_index=menu('Choose the IDA results file you want to plot',filename.name);
eval(['load  ',filename(filename_index).name]);

stride=menu('Do you want to analyze stance or stride?','stride','stance');
if stride==1
    tstart= input(' Enter the frame number where IDA analysis for taga starts: ');
    tstancestop=input(' Enter the frame number where the stance phase stops for taga: ');
    tstop=input(' Enter the frame number where IDA analysis for taga stops: ');
elseif stride==2
    tstart= input(' Enter the frame number where IDA analysis for taga starts: ');
    tstancestop=input(' Enter the frame number where the stance phase stops for taga: ');
    tstop=tstancestop;
end

%====================== Calculating the reconstruction ==============================
% Note: For the IDA data: Ankle and hip angular acceleration is extension positive and knee and
lumbar acceleration is flexion positive.
%       Also pelvis rotation is anticlockwise positive.
% Note: For the cosim data (actual): Ankle and knee angular acceleration are extension positive,
%       Hip and lumbar angular acceleration are flexion positive. Pelvis rotation is
anticlockwise positive.
ti=tstart:tstancestop; % stance interval for the GRF reconstruction
CAL_tRGRF(ti,1)=tRGRF_vel(ti,1)+tRGRF_Lumbar(ti,1)+tRGRF_RHIP(ti,1)+tRGRF_RKNEE(ti,1)+tRGRF_RANKL
E(ti,1)+tRGRF_LHIP(ti,1)+tRGRF_LKNEE(ti,1)+tRGRF_LANKLE(ti,1)+tRGRF_GRAV(ti,1);
CAL_tRGRF(ti,2)=tRGRF_vel(ti,2)+tRGRF_Lumbar(ti,2)+tRGRF_RHIP(ti,2)+tRGRF_RKNEE(ti,2)+tRGRF_RANKL
E(ti,2)+tRGRF_LHIP(ti,2)+tRGRF_LKNEE(ti,2)+tRGRF_LANKLE(ti,2)+tRGRF_GRAV(ti,2);
ti=tstart:tstop; % stride interval for the acceleration reconstruction.
CAL_tRAacc(ti,3)=tRAacc_vel(ti,3)+tRAacc_Lumbar(ti,3)+tRAacc_RHIP(ti,3)+tRAacc_RKNEE(ti,3)+tRAacc
_RANKLE(ti,3)+tRAacc_LHIP(ti,3)+tRAacc_LKNEE(ti,3)+tRAacc_LANKLE(ti,3)+tRAacc_GRAV(ti,3);
CAL_tRKacc(ti,3)=tRKacc_vel(ti,3)+tRKacc_Lumbar(ti,3)+tRKacc_RHIP(ti,3)+tRKacc_RKNEE(ti,3)+tRKacc
_RANKLE(ti,3)+tRKacc_LHIP(ti,3)+tRKacc_LKNEE(ti,3)+tRKacc_LANKLE(ti,3)+tRKacc_GRAV(ti,3);
CAL_tRHacc(ti,3)=tRHacc_vel(ti,3)+tRHacc_Lumbar(ti,3)+tRHacc_RHIP(ti,3)+tRHacc_RKNEE(ti,3)+tRHacc
_RANKLE(ti,3)+tRHacc_LHIP(ti,3)+tRHacc_LKNEE(ti,3)+tRHacc_LANKLE(ti,3)+tRHacc_GRAV(ti,3);
CAL_tLAacc(ti,3)=tLAacc_vel(ti,3)+tLAacc_Lumbar(ti,3)+tLAacc_RHIP(ti,3)+tLAacc_RKNEE(ti,3)+tLAacc
_RANKLE(ti,3)+tLAacc_LHIP(ti,3)+tLAacc_LKNEE(ti,3)+tLAacc_LANKLE(ti,3)+tLAacc_GRAV(ti,3);
CAL_tLKacc(ti,3)=tLKacc_vel(ti,3)+tLKacc_Lumbar(ti,3)+tLKacc_RHIP(ti,3)+tLKacc_RKNEE(ti,3)+tLKacc
_RANKLE(ti,3)+tLKacc_LHIP(ti,3)+tLKacc_LKNEE(ti,3)+tLKacc_LANKLE(ti,3)+tLKacc_GRAV(ti,3);
CAL_tLHacc(ti,3)=tLHacc_vel(ti,3)+tLHacc_Lumbar(ti,3)+tLHacc_RHIP(ti,3)+tLHacc_RKNEE(ti,3)+tLHacc
_RANKLE(ti,3)+tLHacc_LHIP(ti,3)+tLHacc_LKNEE(ti,3)+tLHacc_LANKLE(ti,3)+tLHacc_GRAV(ti,3);
CAL_tPELVISacc(ti,:)=tPELVISacc_vel(ti,:)+tPELVISacc_Lumbar(ti,:)+tPELVISacc_RHIP(ti,:)+tPELVISac
c_RKNEE(ti,:)+tPELVISacc_RANKLE(ti,:)+tPELVISacc_LHIP(ti,:)+tPELVISacc_LKNEE(ti,:)+tPELVISacc_LAN
KLE(ti,:)+tPELVISacc_GRAV(ti,:);
CAL_tLUMacc(ti,3)=tLUMacc_vel(ti,3)+tLUMacc_Lumbar(ti,3)+tLUMacc_RHIP(ti,3)+tLUMacc_RKNEE(ti,3)+t
LUMacc_RANKLE(ti,3)+tLUMacc_LHIP(ti,3)+tLUMacc_LKNEE(ti,3)+tLUMacc_LANKLE(ti,3)+tLUMacc_GRAV(ti,3
);


% calculating the error term
ti=tstart:tstancestop;% stance interval for the GRF reconstruction.
tRGRF_err(ti,1)=RGRF(ti,1)-CAL_tRGRF(ti,1);
tRGRF_err(ti,2)=RGRF(ti,2)-CAL_tRGRF(ti,2);
ti=tstart:tstop; % stride interval for the acceleration reconstruction.
tRAacc_err(ti,3)=RAacc(ti,3)-CAL_tRAacc(ti,3);
tRKacc_err(ti,3)=RKacc(ti,3)-CAL_tRKacc(ti,3);
tRHacc_err(ti,3)=RHacc(ti,3)-CAL_tRHacc(ti,3);
```

```
tLAacc_err(ti,3)=LAacc(ti,3)-CAL_tLAacc(ti,3);
tLKacc_err(ti,3)=LKacc(ti,3)-CAL_tLKacc(ti,3); tLHacc_err(ti,3)=LHacc(ti,3)-CAL_tLHacc(ti,3);
tPELVISacc_err(ti,:)=PELVISacc(ti,:)-CAL_tPELVISacc(ti,:);
tLUMacc_err(ti,3)=LUMacc(ti,3)-CAL_tLUMacc(ti,3);

% Error weighting matrices, Wc & Wgv - similar in function to the C (PI) coefficient matrix...
% This will distribute the 'error torque' according to the 'weights' of the calculated source
loads in the IDA.
% Although the sum of these 'weights' is zero, because the torques have both positive and
negative values, some of the weights may be greater than 1.
% There is a W matrix for both the C & GV matrices
% Since A = C*T + GV + E  (Note: GEV = GV + E, all [10,1,time] vectors, representing
accelerations)
% If we assume that the error (in an acceleration reconstruction) is directly related to the
contributions of the source loads in that recontruction,
% we will distribute the error among all source loads, thus eliminating the separate error term.
for i=tstart:tstop
    Wc(:,:,i)=[tLUMacc_Lumbar(i,3)/CAL_tLUMacc(i,3)  tLUMacc_RHIP(i,3)/CAL_tLUMacc(i,3)
    tLUMacc_LHIP(i,3)/CAL_tLUMacc(i,3)    tLUMacc_RKNEE(i,3)/CAL_tLUMacc(i,3)
    tLUMacc_LKNEE(i,3)/CAL_tLUMacc(i,3)   tLUMacc_RANKLE(i,3)/CAL_tLUMacc(i,3)
    tLUMacc_LANKLE(i,3)/CAL_tLUMacc(i,3)
        tRHacc_Lumbar(i,3)/CAL_tRHacc(i,3) tRHacc_RHIP(i,3)/CAL_tRHacc(i,3)
tRHacc_LHIP(i,3)/CAL_tRHacc(i,3) tRHacc_RKNEE(i,3)/CAL_tRHacc(i,3)
tRHacc_LKNEE(i,3)/CAL_tRHacc(i,3) tRHacc_RANKLE(i,3)/CAL_tRHacc(i,3)
tRHacc_LANKLE(i,3)/CAL_tRHacc(i,3)
        tRKacc_Lumbar(i,3)/CAL_tRKacc(i,3) tRKacc_RHIP(i,3)/CAL_tRKacc(i,3)
tRKacc_LHIP(i,3)/CAL_tRKacc(i,3) tRKacc_RKNEE(i,3)/CAL_tRKacc(i,3)
tRKacc_LKNEE(i,3)/CAL_tRKacc(i,3) tRKacc_RANKLE(i,3)/CAL_tRKacc(i,3)
tRKacc_LANKLE(i,3)/CAL_tRKacc(i,3)
        tRAacc_Lumbar(i,3)/CAL_tRAacc(i,3) tRAacc_RHIP(i,3)/CAL_tRAacc(i,3)
tRAacc_LHIP(i,3)/CAL_tRAacc(i,3) tRAacc_RKNEE(i,3)/CAL_tRAacc(i,3)
tRAacc_LKNEE(i,3)/CAL_tRAacc(i,3) tRAacc_RANKLE(i,3)/CAL_tRAacc(i,3)
tRAacc_LANKLE(i,3)/CAL_tRAacc(i,3)
        tLHacc_Lumbar(i,3)/CAL_tLHacc(i,3) tLHacc_RHIP(i,3)/CAL_tLHacc(i,3)
tLHacc_LHIP(i,3)/CAL_tLHacc(i,3) tLHacc_RKNEE(i,3)/CAL_tLHacc(i,3)
tLHacc_LKNEE(i,3)/CAL_tLHacc(i,3) tLHacc_RANKLE(i,3)/CAL_tLHacc(i,3)
tLHacc_LANKLE(i,3)/CAL_tLHacc(i,3)
        tLKacc_Lumbar(i,3)/CAL_tLKacc(i,3) tLKacc_RHIP(i,3)/CAL_tLKacc(i,3)
tLKacc_LHIP(i,3)/CAL_tLKacc(i,3) tLKacc_RKNEE(i,3)/CAL_tLKacc(i,3)
tLKacc_LKNEE(i,3)/CAL_tLKacc(i,3) tLKacc_RANKLE(i,3)/CAL_tLKacc(i,3)
tLKacc_LANKLE(i,3)/CAL_tLKacc(i,3)
        tLAacc_Lumbar(i,3)/CAL_tLAacc(i,3) tLAacc_RHIP(i,3)/CAL_tLAacc(i,3)
tLAacc_LHIP(i,3)/CAL_tLAacc(i,3) tLAacc_RKNEE(i,3)/CAL_tLAacc(i,3)
tLAacc_LKNEE(i,3)/CAL_tLAacc(i,3) tLAacc_RANKLE(i,3)/CAL_tLAacc(i,3)
tLAacc_LANKLE(i,3)/CAL_tLAacc(i,3)
        tPELVISacc_Lumbar(i,1)/CAL_tPELVISacc(i,1) tPELVISacc_RHIP(i,1)/CAL_tPELVISacc(i,1)
tPELVISacc_LHIP(i,1)/CAL_tPELVISacc(i,1) tPELVISacc_RKNEE(i,1)/CAL_tPELVISacc(i,1)
tPELVISacc_LKNEE(i,1)/CAL_tPELVISacc(i,1) tPELVISacc_RANKLE(i,1)/CAL_tPELVISacc(i,1)
tPELVISacc_LANKLE(i,1)/CAL_tPELVISacc(i,1)
        tPELVISacc_Lumbar(i,2)/CAL_tPELVISacc(i,2) tPELVISacc_RHIP(i,2)/CAL_tPELVISacc(i,2)
tPELVISacc_LHIP(i,2)/CAL_tPELVISacc(i,2) tPELVISacc_RKNEE(i,2)/CAL_tPELVISacc(i,2)
tPELVISacc_LKNEE(i,2)/CAL_tPELVISacc(i,2) tPELVISacc_RANKLE(i,2)/CAL_tPELVISacc(i,2)
tPELVISacc_LANKLE(i,2)/CAL_tPELVISacc(i,2)
        tPELVISacc_Lumbar(i,3)/CAL_tPELVISacc(i,3) tPELVISacc_RHIP(i,3)/CAL_tPELVISacc(i,3)
tPELVISacc_LHIP(i,3)/CAL_tPELVISacc(i,3) tPELVISacc_RKNEE(i,3)/CAL_tPELVISacc(i,3)
tPELVISacc_LKNEE(i,3)/CAL_tPELVISacc(i,3) tPELVISacc_RANKLE(i,3)/CAL_tPELVISacc(i,3)
tPELVISacc_LANKLE(i,3)/CAL_tPELVISacc(i,3)];

    Wgv(:,:,i)=[tLUMacc_vel(i,3)/CAL_tLUMacc(i,3) tLUMacc_GRAV(i,3)/CAL_tLUMacc(i,3)
        tRHacc_vel(i,3)/CAL_tRHacc(i,3) tRHacc_GRAV(i,3)/CAL_tRHacc(i,3)
        tRKacc_vel(i,3)/CAL_tRKacc(i,3) tRKacc_GRAV(i,3)/CAL_tRKacc(i,3)
        tRAacc_vel(i,3)/CAL_tRAacc(i,3) tRAacc_GRAV(i,3)/CAL_tRAacc(i,3)
        tLHacc_vel(i,3)/CAL_tLHacc(i,3) tLHacc_GRAV(i,3)/CAL_tLHacc(i,3)
        tLKacc_vel(i,3)/CAL_tLKacc(i,3) tLKacc_GRAV(i,3)/CAL_tLKacc(i,3)
        tLAacc_vel(i,3)/CAL_tLAacc(i,3) tLAacc_GRAV(i,3)/CAL_tLAacc(i,3)
        tPELVISacc_vel(i,1)/CAL_tPELVISacc(i,1) tPELVISacc_GRAV(i,1)/CAL_tPELVISacc(i,1)
        tPELVISacc_vel(i,2)/CAL_tPELVISacc(i,2) tPELVISacc_GRAV(i,2)/CAL_tPELVISacc(i,2)
        tPELVISacc_vel(i,3)/CAL_tPELVISacc(i,3) tPELVISacc_GRAV(i,3)/CAL_tPELVISacc(i,3)];
end

%========== Calculating the "positional inertia" values for each source load at each time
instant for the BASELINE WALK ============================
```

```
% Note: No use to find the positional inertia values for velocity, GRAV, error as they don't
change.
% Our assumption is that the body position and orientation does not change after weakness.

T=Ta+Tp; % joint torque, extension +ve.
for ti=tstart:tstop

    E(:,1,ti)=[tLUMacc_err(ti,3)
        tRHacc_err(ti,3)
        tRKacc_err(ti,3)
        tRAacc_err(ti,3)
        tLHacc_err(ti,3)
        tLKacc_err(ti,3)
        tLAacc_err(ti,3)
        tPELVISacc_err(ti,1)
        tPELVISacc_err(ti,2)
        tPELVISacc_err(ti,3)];

    %LUMBAR CONTRIBUTION==============
    PI_tLUMacc_Lumbar(ti,3)=(tLUMacc_Lumbar(ti,3))./T(ti,1);
    PI_tRHacc_Lumbar(ti,3)=(tRHacc_Lumbar(ti,3))./T(ti,1);
    PI_tRKacc_Lumbar(ti,3)=(tRKacc_Lumbar(ti,3))./T(ti,1);
    PI_tRAacc_Lumbar(ti,3)=(tRAacc_Lumbar(ti,3))./T(ti,1);
    PI_tLHacc_Lumbar(ti,3)=(tLHacc_Lumbar(ti,3))./T(ti,1);
    PI_tLKacc_Lumbar(ti,3)=(tLKacc_Lumbar(ti,3))./T(ti,1);
    PI_tLAacc_Lumbar(ti,3)=(tLAacc_Lumbar(ti,3))./T(ti,1);
    PI_tPELVISacc_Lumbar(ti,1)=(tPELVISacc_Lumbar(ti,1))./T(ti,1);
    PI_tPELVISacc_Lumbar(ti,2)=(tPELVISacc_Lumbar(ti,2))./T(ti,1);
    PI_tPELVISacc_Lumbar(ti,3)=(tPELVISacc_Lumbar(ti,3))./T(ti,1);

    ePI_tLUMacc_Lumbar(ti,3)=(tLUMacc_Lumbar(ti,3)+Wc(1,1,ti)*E(1,:,ti))./T(ti,1);
    ePI_tRHacc_Lumbar(ti,3)=(tRHacc_Lumbar(ti,3)+Wc(2,1,ti)*E(2,:,ti))./T(ti,1);
    ePI_tRKacc_Lumbar(ti,3)=(tRKacc_Lumbar(ti,3)+Wc(3,1,ti)*E(3,:,ti))./T(ti,1);
    ePI_tRAacc_Lumbar(ti,3)=(tRAacc_Lumbar(ti,3)+Wc(4,1,ti)*E(4,:,ti))./T(ti,1);
    ePI_tLHacc_Lumbar(ti,3)=(tLHacc_Lumbar(ti,3)+Wc(5,1,ti)*E(5,:,ti))./T(ti,1);
    ePI_tLKacc_Lumbar(ti,3)=(tLKacc_Lumbar(ti,3)+Wc(6,1,ti)*E(6,:,ti))./T(ti,1);
    ePI_tLAacc_Lumbar(ti,3)=(tLAacc_Lumbar(ti,3)+Wc(7,1,ti)*E(7,:,ti))./T(ti,1);
    ePI_tPELVISacc_Lumbar(ti,1)=(tPELVISacc_Lumbar(ti,1)+Wc(8,1,ti)*E(8,:,ti))./T(ti,1);
    ePI_tPELVISacc_Lumbar(ti,2)=(tPELVISacc_Lumbar(ti,2)+Wc(9,1,ti)*E(9,:,ti))./T(ti,1);
    ePI_tPELVISacc_Lumbar(ti,3)=(tPELVISacc_Lumbar(ti,3)+Wc(10,1,ti)*E(10,:,ti))./T(ti,1);

    % RHIP  CONTRIBUTION============
    PI_tLUMacc_RHIP(ti,3)=(tLUMacc_RHIP(ti,3))./T(ti,2);
    PI_tRHacc_RHIP(ti,3)=(tRHacc_RHIP(ti,3))./T(ti,2);
    PI_tRKacc_RHIP(ti,3)=(tRKacc_RHIP(ti,3))./T(ti,2);
    PI_tRAacc_RHIP(ti,3)=(tRAacc_RHIP(ti,3))./T(ti,2);
    PI_tLHacc_RHIP(ti,3)=(tLHacc_RHIP(ti,3))./T(ti,2);
    PI_tLKacc_RHIP(ti,3)=(tLKacc_RHIP(ti,3))./T(ti,2);
    PI_tLAacc_RHIP(ti,3)=(tLAacc_RHIP(ti,3))./T(ti,2);
    PI_tPELVISacc_RHIP(ti,1)=(tPELVISacc_RHIP(ti,1))./T(ti,2);
    PI_tPELVISacc_RHIP(ti,2)=(tPELVISacc_RHIP(ti,2))./T(ti,2);
    PI_tPELVISacc_RHIP(ti,3)=(tPELVISacc_RHIP(ti,3))./T(ti,2);

    ePI_tLUMacc_RHIP(ti,3)=(tLUMacc_RHIP(ti,3)+Wc(1,2,ti)*E(1,:,ti))./T(ti,2);
    ePI_tRHacc_RHIP(ti,3)=(tRHacc_RHIP(ti,3)+Wc(2,2,ti)*E(2,:,ti))./T(ti,2);
    ePI_tRKacc_RHIP(ti,3)=(tRKacc_RHIP(ti,3)+Wc(3,2,ti)*E(3,:,ti))./T(ti,2);
    ePI_tRAacc_RHIP(ti,3)=(tRAacc_RHIP(ti,3)+Wc(4,2,ti)*E(4,:,ti))./T(ti,2);
    ePI_tLHacc_RHIP(ti,3)=(tLHacc_RHIP(ti,3)+Wc(5,2,ti)*E(5,:,ti))./T(ti,2);
    ePI_tLKacc_RHIP(ti,3)=(tLKacc_RHIP(ti,3)+Wc(6,2,ti)*E(6,:,ti))./T(ti,2);
    ePI_tLAacc_RHIP(ti,3)=(tLAacc_RHIP(ti,3)+Wc(7,2,ti)*E(7,:,ti))./T(ti,2);
    ePI_tPELVISacc_RHIP(ti,1)=(tPELVISacc_RHIP(ti,1)+Wc(8,2,ti)*E(8,:,ti))./T(ti,2);
    ePI_tPELVISacc_RHIP(ti,2)=(tPELVISacc_RHIP(ti,2)+Wc(9,2,ti)*E(9,:,ti))./T(ti,2);
    ePI_tPELVISacc_RHIP(ti,3)=(tPELVISacc_RHIP(ti,3)+Wc(10,2,ti)*E(10,:,ti))./T(ti,2);

    % RKNEE CONTRIBUTION===============
    PI_tLUMacc_RKNEE(ti,3)=(tLUMacc_RKNEE(ti,3))./T(ti,4);
    PI_tRHacc_RKNEE(ti,3)=(tRHacc_RKNEE(ti,3))./T(ti,4);
    PI_tRKacc_RKNEE(ti,3)=(tRKacc_RKNEE(ti,3))./T(ti,4);
    PI_tRAacc_RKNEE(ti,3)=(tRAacc_RKNEE(ti,3))./T(ti,4);
    PI_tLHacc_RKNEE(ti,3)=(tLHacc_RKNEE(ti,3))./T(ti,4);
    PI_tLKacc_RKNEE(ti,3)=(tLKacc_RKNEE(ti,3))./T(ti,4);
    PI_tLAacc_RKNEE(ti,3)=(tLAacc_RKNEE(ti,3))./T(ti,4);
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PI_tPELVISacc_RKNEE(ti,1)=(tPELVISacc_RKNEE(ti,1))./T(ti,4);
PI_tPELVISacc_RKNEE(ti,2)=(tPELVISacc_RKNEE(ti,2))./T(ti,4);
PI_tPELVISacc_RKNEE(ti,3)=(tPELVISacc_RKNEE(ti,3))./T(ti,4);

ePI_tLUMacc_RKNEE(ti,3)=(tLUMacc_RKNEE(ti,3)+Wc(1,4,ti)*E(1,:,ti))./T(ti,4);
ePI_tRHacc_RKNEE(ti,3)=(tRHacc_RKNEE(ti,3)+Wc(2,4,ti)*E(2,:,ti))./T(ti,4);
ePI_tRKacc_RKNEE(ti,3)=(tRKacc_RKNEE(ti,3)+Wc(3,4,ti)*E(3,:,ti))./T(ti,4);
ePI_tRAacc_RKNEE(ti,3)=(tRAacc_RKNEE(ti,3)+Wc(4,4,ti)*E(4,:,ti))./T(ti,4);
ePI_tLHacc_RKNEE(ti,3)=(tLHacc_RKNEE(ti,3)+Wc(5,4,ti)*E(5,:,ti))./T(ti,4);
ePI_tLKacc_RKNEE(ti,3)=(tLKacc_RKNEE(ti,3)+Wc(6,4,ti)*E(6,:,ti))./T(ti,4);
ePI_tLAacc_RKNEE(ti,3)=(tLAacc_RKNEE(ti,3)+Wc(7,4,ti)*E(7,:,ti))./T(ti,4);
ePI_tPELVISacc_RKNEE(ti,1)=(tPELVISacc_RKNEE(ti,1)+Wc(8,4,ti)*E(8,:,ti))./T(ti,4);
ePI_tPELVISacc_RKNEE(ti,2)=(tPELVISacc_RKNEE(ti,2)+Wc(9,4,ti)*E(9,:,ti))./T(ti,4);
ePI_tPELVISacc_RKNEE(ti,3)=(tPELVISacc_RKNEE(ti,3)+Wc(10,4,ti)*E(10,:,ti))./T(ti,4);


% RANKLE CONTRIBUTION===============
PI_tLUMacc_RANKLE(ti,3)=(tLUMacc_RANKLE(ti,3))./T(ti,6);
PI_tRHacc_RANKLE(ti,3)=(tRHacc_RANKLE(ti,3))./T(ti,6);
PI_tRKacc_RANKLE(ti,3)=(tRKacc_RANKLE(ti,3))./T(ti,6);
PI_tRAacc_RANKLE(ti,3)=(tRAacc_RANKLE(ti,3))./T(ti,6);
PI_tLHacc_RANKLE(ti,3)=(tLHacc_RANKLE(ti,3))./T(ti,6);
PI_tLKacc_RANKLE(ti,3)=(tLKacc_RANKLE(ti,3))./T(ti,6);
PI_tLAacc_RANKLE(ti,3)=(tLAacc_RANKLE(ti,3))./T(ti,6);
PI_tPELVISacc_RANKLE(ti,1)=(tPELVISacc_RANKLE(ti,1))./T(ti,6);
PI_tPELVISacc_RANKLE(ti,2)=(tPELVISacc_RANKLE(ti,2))./T(ti,6);
PI_tPELVISacc_RANKLE(ti,3)=(tPELVISacc_RANKLE(ti,3))./T(ti,6);

ePI_tLUMacc_RANKLE(ti,3)=(tLUMacc_RANKLE(ti,3)+Wc(1,6,ti)*E(1,:,ti))./T(ti,6);
ePI_tRHacc_RANKLE(ti,3)=(tRHacc_RANKLE(ti,3)+Wc(2,6,ti)*E(2,:,ti))./T(ti,6);
ePI_tRKacc_RANKLE(ti,3)=(tRKacc_RANKLE(ti,3)+Wc(3,6,ti)*E(3,:,ti))./T(ti,6);
ePI_tRAacc_RANKLE(ti,3)=(tRAacc_RANKLE(ti,3)+Wc(4,6,ti)*E(4,:,ti))./T(ti,6);
ePI_tLHacc_RANKLE(ti,3)=(tLHacc_RANKLE(ti,3)+Wc(5,6,ti)*E(5,:,ti))./T(ti,6);
ePI_tLKacc_RANKLE(ti,3)=(tLKacc_RANKLE(ti,3)+Wc(6,6,ti)*E(6,:,ti))./T(ti,6);
ePI_tLAacc_RANKLE(ti,3)=(tLAacc_RANKLE(ti,3)+Wc(7,6,ti)*E(7,:,ti))./T(ti,6);
ePI_tPELVISacc_RANKLE(ti,1)=(tPELVISacc_RANKLE(ti,1)+Wc(8,6,ti)*E(8,:,ti))./T(ti,6);
ePI_tPELVISacc_RANKLE(ti,2)=(tPELVISacc_RANKLE(ti,2)+Wc(9,6,ti)*E(9,:,ti))./T(ti,6);
ePI_tPELVISacc_RANKLE(ti,3)=(tPELVISacc_RANKLE(ti,3)+Wc(10,6,ti)*E(10,:,ti))./T(ti,6);

% LHIP CONTRIBUTION==================
PI_tLUMacc_LHIP(ti,3)=(tLUMacc_LHIP(ti,3))./T(ti,3);
PI_tRHacc_LHIP(ti,3)=(tRHacc_LHIP(ti,3))./T(ti,3);
PI_tRKacc_LHIP(ti,3)=(tRKacc_LHIP(ti,3))./T(ti,3);
PI_tRAacc_LHIP(ti,3)=(tRAacc_LHIP(ti,3))./T(ti,3);
PI_tLHacc_LHIP(ti,3)=(tLHacc_LHIP(ti,3))./T(ti,3);
PI_tLKacc_LHIP(ti,3)=(tLKacc_LHIP(ti,3))./T(ti,3);
PI_tLAacc_LHIP(ti,3)=(tLAacc_LHIP(ti,3))./T(ti,3);
PI_tPELVISacc_LHIP(ti,1)=(tPELVISacc_LHIP(ti,1))./T(ti,3);
PI_tPELVISacc_LHIP(ti,2)=(tPELVISacc_LHIP(ti,2))./T(ti,3);
PI_tPELVISacc_LHIP(ti,3)=(tPELVISacc_LHIP(ti,3))./T(ti,3);

ePI_tLUMacc_LHIP(ti,3)=(tLUMacc_LHIP(ti,3)+Wc(1,3,ti)*E(1,:,ti))./T(ti,3);
ePI_tRHacc_LHIP(ti,3)=(tRHacc_LHIP(ti,3)+Wc(2,3,ti)*E(2,:,ti))./T(ti,3);
ePI_tRKacc_LHIP(ti,3)=(tRKacc_LHIP(ti,3)+Wc(3,3,ti)*E(3,:,ti))./T(ti,3);
ePI_tRAacc_LHIP(ti,3)=(tRAacc_LHIP(ti,3)+Wc(4,3,ti)*E(4,:,ti))./T(ti,3);
ePI_tLHacc_LHIP(ti,3)=(tLHacc_LHIP(ti,3)+Wc(5,3,ti)*E(5,:,ti))./T(ti,3);
ePI_tLKacc_LHIP(ti,3)=(tLKacc_LHIP(ti,3)+Wc(6,3,ti)*E(6,:,ti))./T(ti,3);
ePI_tLAacc_LHIP(ti,3)=(tLAacc_LHIP(ti,3)+Wc(7,3,ti)*E(7,:,ti))./T(ti,3);
ePI_tPELVISacc_LHIP(ti,1)=(tPELVISacc_LHIP(ti,1)+Wc(8,3,ti)*E(8,:,ti))./T(ti,3);
ePI_tPELVISacc_LHIP(ti,2)=(tPELVISacc_LHIP(ti,2)+Wc(9,3,ti)*E(9,:,ti))./T(ti,3);
ePI_tPELVISacc_LHIP(ti,3)=(tPELVISacc_LHIP(ti,3)+Wc(10,3,ti)*E(10,:,ti))./T(ti,3);


% LKNEE CONTRIBUTION===============
PI_tLUMacc_LKNEE(ti,3)=(tLUMacc_LKNEE(ti,3))./T(ti,5);
PI_tRHacc_LKNEE(ti,3)=(tRHacc_LKNEE(ti,3))./T(ti,5);
PI_tRKacc_LKNEE(ti,3)=(tRKacc_LKNEE(ti,3))./T(ti,5);
PI_tRAacc_LKNEE(ti,3)=(tRAacc_LKNEE(ti,3))./T(ti,5);
PI_tLHacc_LKNEE(ti,3)=(tLHacc_LKNEE(ti,3))./T(ti,5);
PI_tLKacc_LKNEE(ti,3)=(tLKacc_LKNEE(ti,3))./T(ti,5);
PI_tLAacc_LKNEE(ti,3)=(tLAacc_LKNEE(ti,3))./T(ti,5);
PI_tPELVISacc_LKNEE(ti,1)=(tPELVISacc_LKNEE(ti,1))./T(ti,5);
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    PI_tPELVISacc_LKNEE(ti,2)=(tPELVISacc_LKNEE(ti,2))./T(ti,5);
    PI_tPELVISacc_LKNEE(ti,3)=(tPELVISacc_LKNEE(ti,3))./T(ti,5);

    ePI_tLUMacc_LKNEE(ti,3)=(tLUMacc_LKNEE(ti,3)+Wc(1,5,ti)*E(1,:,ti))./T(ti,5);
    ePI_tRHacc_LKNEE(ti,3)=(tRHacc_LKNEE(ti,3)+Wc(2,5,ti)*E(2,:,ti))./T(ti,5);
    ePI_tRKacc_LKNEE(ti,3)=(tRKacc_LKNEE(ti,3)+Wc(3,5,ti)*E(3,:,ti))./T(ti,5);
    ePI_tRAacc_LKNEE(ti,3)=(tRAacc_LKNEE(ti,3)+Wc(4,5,ti)*E(4,:,ti))./T(ti,5);
    ePI_tLHacc_LKNEE(ti,3)=(tLHacc_LKNEE(ti,3)+Wc(5,5,ti)*E(5,:,ti))./T(ti,5);
    ePI_tLKacc_LKNEE(ti,3)=(tLKacc_LKNEE(ti,3)+Wc(6,5,ti)*E(6,:,ti))./T(ti,5);
    ePI_tLAacc_LKNEE(ti,3)=(tLAacc_LKNEE(ti,3)+Wc(7,5,ti)*E(7,:,ti))./T(ti,5);
    ePI_tPELVISacc_LKNEE(ti,1)=(tPELVISacc_LKNEE(ti,1)+Wc(8,5,ti)*E(8,:,ti))./T(ti,5);
    ePI_tPELVISacc_LKNEE(ti,2)=(tPELVISacc_LKNEE(ti,2)+Wc(9,5,ti)*E(9,:,ti))./T(ti,5);
    ePI_tPELVISacc_LKNEE(ti,3)=(tPELVISacc_LKNEE(ti,3)+Wc(10,5,ti)*E(10,:,ti))./T(ti,5);

    % LANKLE CONTRIBUTION=================
    PI_tLUMacc_LANKLE(ti,3)=(tLUMacc_LANKLE(ti,3))./T(ti,7);
    PI_tRHacc_LANKLE(ti,3)=(tRHacc_LANKLE(ti,3))./T(ti,7);
    PI_tRKacc_LANKLE(ti,3)=(tRKacc_LANKLE(ti,3))./T(ti,7);
    PI_tRAacc_LANKLE(ti,3)=(tRAacc_LANKLE(ti,3))./T(ti,7);
    PI_tLHacc_LANKLE(ti,3)=(tLHacc_LANKLE(ti,3))./T(ti,7);
    PI_tLKacc_LANKLE(ti,3)=(tLKacc_LANKLE(ti,3))./T(ti,7);
    PI_tLAacc_LANKLE(ti,3)=(tLAacc_LANKLE(ti,3))./T(ti,7);
    PI_tPELVISacc_LANKLE(ti,1)=(tPELVISacc_LANKLE(ti,1))./T(ti,7);
    PI_tPELVISacc_LANKLE(ti,2)=(tPELVISacc_LANKLE(ti,2))./T(ti,7);
    PI_tPELVISacc_LANKLE(ti,3)=(tPELVISacc_LANKLE(ti,3))./T(ti,7);

    ePI_tLUMacc_LANKLE(ti,3)=(tLUMacc_LANKLE(ti,3)+Wc(1,7,ti)*E(1,:,ti))./T(ti,7);
    ePI_tRHacc_LANKLE(ti,3)=(tRHacc_LANKLE(ti,3)+Wc(2,7,ti)*E(2,:,ti))./T(ti,7);
    ePI_tRKacc_LANKLE(ti,3)=(tRKacc_LANKLE(ti,3)+Wc(3,7,ti)*E(3,:,ti))./T(ti,7);
    ePI_tRAacc_LANKLE(ti,3)=(tRAacc_LANKLE(ti,3)+Wc(4,7,ti)*E(4,:,ti))./T(ti,7);
    ePI_tLHacc_LANKLE(ti,3)=(tLHacc_LANKLE(ti,3)+Wc(5,7,ti)*E(5,:,ti))./T(ti,7);
    ePI_tLKacc_LANKLE(ti,3)=(tLKacc_LANKLE(ti,3)+Wc(6,7,ti)*E(6,:,ti))./T(ti,7);
    ePI_tLAacc_LANKLE(ti,3)=(tLAacc_LANKLE(ti,3)+Wc(7,7,ti)*E(7,:,ti))./T(ti,7);
    ePI_tPELVISacc_LANKLE(ti,1)=(tPELVISacc_LANKLE(ti,1)+Wc(8,7,ti)*E(8,:,ti))./T(ti,7);
    ePI_tPELVISacc_LANKLE(ti,2)=(tPELVISacc_LANKLE(ti,2)+Wc(9,7,ti)*E(9,:,ti))./T(ti,7);
    ePI_tPELVISacc_LANKLE(ti,3)=(tPELVISacc_LANKLE(ti,3)+Wc(10,7,ti)*E(10,:,ti))./T(ti,7);

end

% For the following matrices the third dimension is time (frame number).
% C[i,j,time] = 3D matrix of PI's; i refers to the joint/segment, j to the joint contributors
% eC[i,j,time] is the error weighted ePI* based C matrix.
% GEV[i,1,time] = 3D column matrix of summations of GRAV, err, and vel acceleration components
% GV[i,1,time] is GEV without the error term.
% A[i,1,time] = 3D column matrix of joint/segment net accelerations, i refers to the
joint/segment
% C, GEV & A are based on the non-weakened torques, T, and the following equations are true:
% Eq.1:  A(:,:,time) = C(:,:,time) * T(:,time) + GEV(:,:,time);  and thus
% Eq.2:  T(:,time)= C(:,:,time) \ (A(:,:,time) - GEV(:,:,time));

ti=tstart:tstop;
for i=ti
    C(:,:,i)=[PI_tLUMacc_Lumbar(i,3) PI_tLUMacc_RHIP(i,3) PI_tLUMacc_LHIP(i,3)
PI_tLUMacc_RKNEE(i,3) PI_tLUMacc_LKNEE(i,3) PI_tLUMacc_RANKLE(i,3) PI_tLUMacc_LANKLE(i,3)
        PI_tRHacc_Lumbar(i,3) PI_tRHacc_RHIP(i,3) PI_tRHacc_LHIP(i,3) PI_tRHacc_RKNEE(i,3)
PI_tRHacc_LKNEE(i,3) PI_tRHacc_RANKLE(i,3) PI_tRHacc_LANKLE(i,3)
        PI_tRKacc_Lumbar(i,3) PI_tRKacc_RHIP(i,3) PI_tRKacc_LHIP(i,3) PI_tRKacc_RKNEE(i,3)
PI_tRKacc_LKNEE(i,3) PI_tRKacc_RANKLE(i,3) PI_tRKacc_LANKLE(i,3)
        PI_tRAacc_Lumbar(i,3) PI_tRAacc_RHIP(i,3) PI_tRAacc_LHIP(i,3) PI_tRAacc_RKNEE(i,3)
PI_tRAacc_LKNEE(i,3) PI_tRAacc_RANKLE(i,3) PI_tRAacc_LANKLE(i,3)
        PI_tLHacc_Lumbar(i,3) PI_tLHacc_RHIP(i,3) PI_tLHacc_LHIP(i,3) PI_tLHacc_RKNEE(i,3)
PI_tLHacc_LKNEE(i,3) PI_tLHacc_RANKLE(i,3) PI_tLHacc_LANKLE(i,3)
        PI_tLKacc_Lumbar(i,3) PI_tLKacc_RHIP(i,3) PI_tLKacc_LHIP(i,3) PI_tLKacc_RKNEE(i,3)
PI_tLKacc_LKNEE(i,3) PI_tLKacc_RANKLE(i,3) PI_tLKacc_LANKLE(i,3)
        PI_tLAacc_Lumbar(i,3) PI_tLAacc_RHIP(i,3) PI_tLAacc_LHIP(i,3) PI_tLAacc_RKNEE(i,3)
PI_tLAacc_LKNEE(i,3) PI_tLAacc_RANKLE(i,3) PI_tLAacc_LANKLE(i,3)
        PI_tPELVISacc_Lumbar(i,1) PI_tPELVISacc_RHIP(i,1) PI_tPELVISacc_LHIP(i,1)
PI_tPELVISacc_RKNEE(i,1) PI_tPELVISacc_LKNEE(i,1) PI_tPELVISacc_RANKLE(i,1)
PI_tPELVISacc_LANKLE(i,1)
        PI_tPELVISacc_Lumbar(i,2) PI_tPELVISacc_RHIP(i,2) PI_tPELVISacc_LHIP(i,2)
PI_tPELVISacc_RKNEE(i,2) PI_tPELVISacc_LKNEE(i,2) PI_tPELVISacc_RANKLE(i,2)
PI_tPELVISacc_LANKLE(i,2)
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        PI_tPELVISacc_Lumbar(i,3) PI_tPELVISacc_RHIP(i,3) PI_tPELVISacc_LHIP(i,3)
PI_tPELVISacc_RKNEE(i,3) PI_tPELVISacc_LKNEE(i,3) PI_tPELVISacc_RANKLE(i,3)
PI_tPELVISacc_LANKLE(i,3)];

    eC(:,:,i)=[ePI_tLUMacc_Lumbar(i,3) ePI_tLUMacc_RHIP(i,3) ePI_tLUMacc_LHIP(i,3)
ePI_tLUMacc_RKNEE(i,3) ePI_tLUMacc_LKNEE(i,3) ePI_tLUMacc_RANKLE(i,3) ePI_tLUMacc_LANKLE(i,3)
        ePI_tRHacc_Lumbar(i,3) ePI_tRHacc_RHIP(i,3) ePI_tRHacc_LHIP(i,3) ePI_tRHacc_RKNEE(i,3)
ePI_tRHacc_LKNEE(i,3) ePI_tRHacc_RANKLE(i,3) ePI_tRHacc_LANKLE(i,3)
        ePI_tRKacc_Lumbar(i,3) ePI_tRKacc_RHIP(i,3) ePI_tRKacc_LHIP(i,3) ePI_tRKacc_RKNEE(i,3)
ePI_tRKacc_LKNEE(i,3) ePI_tRKacc_RANKLE(i,3) ePI_tRKacc_LANKLE(i,3)
        ePI_tRAacc_Lumbar(i,3) ePI_tRAacc_RHIP(i,3) ePI_tRAacc_LHIP(i,3) ePI_tRAacc_RKNEE(i,3)
ePI_tRAacc_LKNEE(i,3) ePI_tRAacc_RANKLE(i,3) ePI_tRAacc_LANKLE(i,3)
        ePI_tLHacc_Lumbar(i,3) ePI_tLHacc_RHIP(i,3) ePI_tLHacc_LHIP(i,3) ePI_tLHacc_RKNEE(i,3)
ePI_tLHacc_LKNEE(i,3) ePI_tLHacc_RANKLE(i,3) ePI_tLHacc_LANKLE(i,3)
        ePI_tLKacc_Lumbar(i,3) ePI_tLKacc_RHIP(i,3) ePI_tLKacc_LHIP(i,3) ePI_tLKacc_RKNEE(i,3)
ePI_tLKacc_LKNEE(i,3) ePI_tLKacc_RANKLE(i,3) ePI_tLKacc_LANKLE(i,3)
        ePI_tLAacc_Lumbar(i,3) ePI_tLAacc_RHIP(i,3) ePI_tLAacc_LHIP(i,3) ePI_tLAacc_RKNEE(i,3)
ePI_tLAacc_LKNEE(i,3) ePI_tLAacc_RANKLE(i,3) ePI_tLAacc_LANKLE(i,3)
        ePI_tPELVISacc_Lumbar(i,1) ePI_tPELVISacc_RHIP(i,1) ePI_tPELVISacc_LHIP(i,1)
ePI_tPELVISacc_RKNEE(i,1) ePI_tPELVISacc_LKNEE(i,1) ePI_tPELVISacc_RANKLE(i,1)
ePI_tPELVISacc_LANKLE(i,1)
        ePI_tPELVISacc_Lumbar(i,2) ePI_tPELVISacc_RHIP(i,2) ePI_tPELVISacc_LHIP(i,2)
ePI_tPELVISacc_RKNEE(i,2) ePI_tPELVISacc_LKNEE(i,2) ePI_tPELVISacc_RANKLE(i,2)
ePI_tPELVISacc_LANKLE(i,2)
        ePI_tPELVISacc_Lumbar(i,3) ePI_tPELVISacc_RHIP(i,3) ePI_tPELVISacc_LHIP(i,3)
ePI_tPELVISacc_RKNEE(i,3) ePI_tPELVISacc_LKNEE(i,3) ePI_tPELVISacc_RANKLE(i,3)
ePI_tPELVISacc_LANKLE(i,3)];

    GV(:,1,i)=[tLUMacc_GRAV(i,3) + tLUMacc_vel(i,3)
        tRHacc_GRAV(i,3)+ tRHacc_vel(i,3)
        tRKacc_GRAV(i,3)+ tRKacc_vel(i,3)
        tRAacc_GRAV(i,3)+ tRAacc_vel(i,3)
        tLHacc_GRAV(i,3)+ tLHacc_vel(i,3)
        tLKacc_GRAV(i,3)+ tLKacc_vel(i,3)
        tLAacc_GRAV(i,3)+ tLAacc_vel(i,3)
        tPELVISacc_GRAV(i,1)+ tPELVISacc_vel(i,1)
        tPELVISacc_GRAV(i,2)+ tPELVISacc_vel(i,2)
        tPELVISacc_GRAV(i,3)+ tPELVISacc_vel(i,3)];

    eGV(:,1,i)=[(tLUMacc_GRAV(i,3)+Wgv(1,2,i)*E(1,:,i)) + (tLUMacc_vel(i,3)+Wgv(1,1,i)*E(1,:,i))
        (tRHacc_GRAV(i,3)+Wgv(2,2,i)*E(2,:,i))+ (tRHacc_vel(i,3)+Wgv(2,1,i)*E(2,:,i))
        (tRKacc_GRAV(i,3)+Wgv(3,2,i)*E(3,:,i))+ (tRKacc_vel(i,3)+Wgv(3,1,i)*E(3,:,i))
        (tRAacc_GRAV(i,3)+Wgv(4,2,i)*E(4,:,i))+ (tRAacc_vel(i,3)+Wgv(4,1,i)*E(4,:,i))
        (tLHacc_GRAV(i,3)+Wgv(5,2,i)*E(5,:,i))+ (tLHacc_vel(i,3)+Wgv(5,1,i)*E(5,:,i))
        (tLKacc_GRAV(i,3)+Wgv(6,2,i)*E(6,:,i))+ (tLKacc_vel(i,3)+Wgv(6,1,i)*E(6,:,i))
        (tLAacc_GRAV(i,3)+Wgv(7,2,i)*E(7,:,i))+ (tLAacc_vel(i,3)+Wgv(7,1,i)*E(7,:,i))
        (tPELVISacc_GRAV(i,1)+Wgv(8,2,i)*E(8,:,i))+ (tPELVISacc_vel(i,1)+Wgv(8,1,i)*E(8,:,i))
        (tPELVISacc_GRAV(i,2)+Wgv(9,2,i)*E(9,:,i))+ (tPELVISacc_vel(i,2)+Wgv(9,1,i)*E(9,:,i))
        (tPELVISacc_GRAV(i,3)+Wgv(10,2,i)*E(10,:,i))+
(tPELVISacc_vel(i,3)+Wgv(10,1,i)*E(10,:,i))];

    A(:,1,i)=[LUMacc(i,3); RHacc(i,3); RKacc(i,3); RAacc(i,3); LHacc(i,3); LKacc(i,3);
LAacc(i,3); PELVISacc(i,1); PELVISacc(i,2); PELVISacc(i,3)];
end

GEV=GV+E;

% To run the LSQLIN method, need to divide the coefficient matrix C into 2 subset matrices F &
Aeq,
% and the acceleration vector A into subvectors d & beq. LSQLIN minimizes F*x-d such that
Aeq*x==beq.
% This section chooses which accelerations are constrained in Aeq & beq.
ACCeval=strvcat('Choose LSQLIN accelerations to constrain.','When finished choosing, select
Done.','None selected');
ACClist=strvcat('Lumbar','RHip','RKnee','RAnkle','LHip','LKnee','LAnkle','Pelvis x','Pelvis
y','Pelvis rot','Done');
for i=1:11
    ACC(i).name=ACClist(i,:);
end
ACCcho=0; cnt=1;
while ACCcho<11
    ACCcho(cnt)=menu(ACCeval,ACC.name);
```

```
    ACCeval=strvcat(ACCeval(1,:),ACCeval(2,:),'Accelerations selected:',char(ACC(ACCcho).name));
    cnt=cnt+1;
end
allset=[1:10];                  % LSQcho & CONcho are indices for the LSQLIN function
allset(ACCcho(1:end-1))=0;      % This method eliminates repeated selections
LSQcho=find(allset)             % LSQ or lsq: LSQ unconstrained portion, i.e.: [3 5 6 7 8 9 10]
allset=[1:10];
allset(LSQcho)=0;
CONcho=find(allset);            % CON or con: CONstrained portion, i.e.: [1 2 4], complement to
LSQcho

%Clsq=C(LSQcho,:,:);
%Ccon=C(CONcho,:,:);
GEVlsq=GEV(LSQcho,:,:);
GEVcon=GEV(CONcho,:,:);
Alsq=A(LSQcho,:,:);
Acon=A(CONcho,:,:);

%eClsq=eC(LSQcho,:,:);
%eCcon=eC(CONcho,:,:);
eGVlsq=eGV(LSQcho,:,:);
eGVcon=eGV(CONcho,:,:);

%========================= Introduction of the weakness  ================================
% To introduce the weakness in the muscle, calculate the joint torque due to the weakened muscle,
% calculate the joint moment contribution of the weakened torque and then calculate the
% compensation scheme.

weak=menu('Do you want to introduce weakness?','Yes','No');
if weak==1
    % Calculation of weakened T values...
    % default is full strength for all the muscles...
    % hip extensors
    stren_Tm4=1;
    stren_Tm6=1;
    stren_Tm8=1;
    stren_Tm10=1;
    % knee extensors
    stren_Tm7=1;
    stren_Tm9=1;
    stren_Tm12=1;
    stren_Tm14=1;
    % ankle extensors
    stren_Tm19=1;
    stren_Tm16=1;
    stren_Tm18=1;
    stren_Tm20=1;

    weak_grp=menu('Which group of muscles due you want to weaken?','Hip Extensors','Knee
Extensors','Ankle Extensors');
    side=menu('Which side do you want to weaken?','right','left');
    if weak_grp==1 % weak hip extensors
        hip_ext=input(' Enter the strength of the hip extensors as a fraction. (1 for full
strength and 0 for no strength): ');
        if side==1 % right
            stren_Tm4=hip_ext;
            stren_Tm8=hip_ext;
            name='Right Hip extensors';
            wkname=['RHe',int2str(hip_ext*100)];
        elseif side==2 % left
            stren_Tm6=hip_ext;
            stren_Tm10=hip_ext;
            name='Left Hip extensors';
            wkname=['LHe',int2str(hip_ext*100)];
        end
    end
    if weak_grp==2 % weak knee extensors
        knee_ext=input(' Enter the strength of the knee extensors as a fraction. (1 for full
strength and 0 for no strength): ');
        if side==1 % right
            stren_Tm7=knee_ext;
            stren_Tm12=knee_ext;
```

```
            name='Right Knee extensors';
            wkname=['RKe',int2str(knee_ext*100)];
        elseif side==2 % left
            stren_Tm9=knee_ext;
            stren_Tm14=knee_ext;
            name='Left Knee extensors';
            wkname=['LHe',int2str(knee_ext*100)];
        end
    end
    if weak_grp==3 % weak ankle extensors
        ankle_ext=input(' Enter the strength of the ankle extensors as a fraction. (1 for full
strength and 0 for no strength): ');
        if side==1 % right
            stren_Tm16=ankle_ext;
            stren_Tm19=ankle_ext;
            name='Right Ankle extensors';
            wkname=['RAe',int2str(ankle_ext*100)];
        elseif side==2 %left
            stren_Tm18=ankle_ext;
            stren_Tm20=ankle_ext;
            name='Left Ankle extensors';
            wkname=['RAe',int2str(ankle_ext*100)];
        end
    end
    % Taw & Tw are the 'weakened' values of Ta & T, extension +ve.
    ti=tstart:tstop;
    Taw(ti,1)=Tm(ti,2)-Tm(ti,1);
    Taw(ti,2)=stren_Tm4*Tm(ti,4)-Tm(ti,3)+stren_Tm8*Tm(ti,8)-Tm(ti,7);
    Taw(ti,3)=stren_Tm6*Tm(ti,6)-Tm(ti,5)+stren_Tm10*Tm(ti,10)-Tm(ti,9);
    Taw(ti,4)=stren_Tm12*Tm(ti,12)-Tm(ti,11)+stren_Tm7*Tm(ti,7)-0.5*Tm(ti,8)-Tm(ti,19);
    Taw(ti,5)=stren_Tm14*Tm(ti,14)-Tm(ti,13)+stren_Tm9*Tm(ti,9)-0.5*Tm(ti,10)-Tm(ti,20);
    Taw(ti,6)=stren_Tm16*Tm(ti,16)-Tm(ti,15)+stren_Tm19*Tm(ti,19);
    Taw(ti,7)=stren_Tm18*Tm(ti,18)-Tm(ti,17)+stren_Tm20*Tm(ti,20);
    Tw(ti,:)=Taw(ti,:)+Tp(ti,:);

    T=T'; Tw=Tw'; % for the torques, the columns now represent the frame number.

    % Choosing the constraints for the contrained least squares method (LINLSQ)
    %

    % For the weakened torques, the weakened joint contributions will remain
    % fixed, and must be separated from the right hand side of Eq.1. Ccomp & Tcomp
    % refer to the non-weakened joints, Cweak & Tweak refer to the weakened joint.
    % These accelerations will be combined with the GEV term in Eq.2.
    % Eq.1a:  A(:,:,time) = Ccomp(:,:,time) * Tcomp(:,time) + Cweak(:,:,time) * Tweak(:,time) +
GEV(:,:,time);  and thus
    % Eq.2a:  Tcomp(:,time)= Ccomp(:,:,time) \ (A(:,:,time) - Cweak(:,:,time) * Tweak(:,time) -
GEV(:,:,time));
    % Cclsq (comp), Cwlsq (weak), Cccon (comp) & Cwcon (weak) are the coefficient matrices for
the LSQLIN method.

    if weak_grp==1 & side==1 % right hip extensors are weak
        Ccomp(:,1:6,:)=[C(:,1,:) C(:,3:7,:)];
        Cweak(:,1,:)=C(:,2,:);
        Tweak(1,:)=Tw(2,:);
        Cclsq(1:size(LSQcho,2),1:6,:)=[C(LSQcho,1,:) C(LSQcho,3:7,:)];
        Cwlsq(1:size(LSQcho,2),1,:)=C(LSQcho,2,:);
        Cccon(1:size(CONcho,2),1:6,:)=[C(CONcho,1,:) C(CONcho,3:7,:)];
        Cwcon(1:size(CONcho,2),1,:)=C(CONcho,2,:);

        eCcomp(:,1:6,:)=[eC(:,1,:) eC(:,3:7,:)];
        eCweak(:,1,:)=eC(:,2,:);
        eCclsq(1:size(LSQcho,2),1:6,:)=[eC(LSQcho,1,:) eC(LSQcho,3:7,:)];
        eCwlsq(1:size(LSQcho,2),1,:)=eC(LSQcho,2,:);
        eCccon(1:size(CONcho,2),1:6,:)=[eC(CONcho,1,:) eC(CONcho,3:7,:)];
        eCwcon(1:size(CONcho,2),1,:)=eC(CONcho,2,:);
    elseif weak_grp==1 & side==2 % left hip extensors are weak
        Ccomp(:,1:6,:)=[C(:,1:2,:) C(:,4:7,:)];
        Cweak(:,1,:)=C(:,3,:);
        Tweak(1,:)=Tw(3,:);
        Cclsq(1:size(LSQcho,2),1:6,:)=[C(LSQcho,1:2,:) C(LSQcho,4:7,:)];
        Cwlsq(1:size(LSQcho,2),1,:)=C(LSQcho,3,:);
```

```
    Cccon(1:size(CONcho,2),1:6,:)=[C(CONcho,1:2,:) C(CONcho,4:7,:)];
    Cwcon(1:size(CONcho,2),1,:)=C(CONcho,3,:);

    eCcomp(:,1:6,:)=[eC(:,1:2,:) eC(:,4:7,:)];
    eCweak(:,1,:)=eC(:,3,:);
    eCclsq(1:size(LSQcho,2),1:6,:)=[eC(LSQcho,1:2,:) eC(LSQcho,4:7,:)];
    eCwlsq(1:size(LSQcho,2),1,:)=eC(LSQcho,3,:);
    eCccon(1:size(CONcho,2),1:6,:)=[eC(CONcho,1:2,:) eC(CONcho,4:7,:)];
    eCwcon(1:size(CONcho,2),1,:)=eC(CONcho,3,:);
elseif weak_grp==2 & side==1 % right knee extensors are weak
    Ccomp(:,1:6,:)=[C(:,1:3,:) C(:,5:7,:)];
    Cweak(:,1,:)=C(:,4,:);
    Tweak(1,:)=Tw(4,:);
    Cclsq(1:size(LSQcho,2),1:6,:)=[C(LSQcho,1:3,:) C(LSQcho,5:7,:)];
    Cwlsq(1:size(LSQcho,2),1,:)=C(LSQcho,4,:);
    Cccon(1:size(CONcho,2),1:6,:)=[C(CONcho,1:3,:) C(CONcho,5:7,:)];
    Cwcon(1:size(CONcho,2),1,:)=C(CONcho,4,:);

    eCcomp(:,1:6,:)=[eC(:,1:3,:) eC(:,5:7,:)];
    eCweak(:,1,:)=eC(:,4,:);
    eCclsq(1:size(LSQcho,2),1:6,:)=[eC(LSQcho,1:3,:) eC(LSQcho,5:7,:)];
    eCwlsq(1:size(LSQcho,2),1,:)=eC(LSQcho,4,:);
    eCccon(1:size(CONcho,2),1:6,:)=[eC(CONcho,1:3,:) eC(CONcho,5:7,:)];
    eCwcon(1:size(CONcho,2),1,:)=eC(CONcho,4,:);
elseif weak_grp==2 & side==2 % left knee extensors are weak
    Ccomp(:,1:6,:)=[C(:,1:4,:) C(:,6:7,:)];
    Cweak(:,1,:)=C(:,5,:);
    Tweak(1,:)=Tw(5,:);
    Cclsq(1:size(LSQcho,2),1:6,:)=[C(LSQcho,1:4,:) C(LSQcho,6:7,:)];
    Cwlsq(1:size(LSQcho,2),1,:)=C(LSQcho,5,:);
    Cccon(1:size(CONcho,2),1:6,:)=[C(CONcho,1:4,:) C(CONcho,6:7,:)];
    Cwcon(1:size(CONcho,2),1,:)=C(CONcho,5,:);

    eCcomp(:,1:6,:)=[eC(:,1:4,:) eC(:,6:7,:)];
    eCweak(:,1,:)=eC(:,5,:);
    eCclsq(1:size(LSQcho,2),1:6,:)=[eC(LSQcho,1:4,:) eC(LSQcho,6:7,:)];
    eCwlsq(1:size(LSQcho,2),1,:)=eC(LSQcho,5,:);
    eCccon(1:size(CONcho,2),1:6,:)=[eC(CONcho,1:4,:) eC(CONcho,6:7,:)];
    eCwcon(1:size(CONcho,2),1,:)=eC(CONcho,5,:);
elseif weak_grp==3 & side==1 % right ankle extensors are weak
    Ccomp(:,1:6,:)=[C(:,1:5,:) C(:,7,:)];
    Cweak(:,1,:)=C(:,6,:);
    Tweak(1,:)=Tw(6,:);
    Cclsq(1:size(LSQcho,2),1:6,:)=[C(LSQcho,1:5,:) C(LSQcho,7,:)];
    Cwlsq(1:size(LSQcho,2),1,:)=C(LSQcho,6,:);
    Cccon(1:size(CONcho,2),1:6,:)=[C(CONcho,1:5,:) C(CONcho,7,:)];
    Cwcon(1:size(CONcho,2),1,:)=C(CONcho,6,:);

    eCcomp(:,1:6,:)=[eC(:,1:5,:) eC(:,7,:)];
    eCweak(:,1,:)=eC(:,6,:);
    eCclsq(1:size(LSQcho,2),1:6,:)=[eC(LSQcho,1:5,:) eC(LSQcho,7,:)];
    eCwlsq(1:size(LSQcho,2),1,:)=eC(LSQcho,6,:);
    eCccon(1:size(CONcho,2),1:6,:)=[eC(CONcho,1:5,:) eC(CONcho,7,:)];
    eCwcon(1:size(CONcho,2),1,:)=eC(CONcho,6,:);

elseif weak_grp==3 & side==2 % left ankle extensors are weak
    Ccomp(:,1:6,:)=C(:,1:6,:);
    Cweak(:,1,:)=C(:,7,:);
    Tweak(1,:)=Tw(7,:);
    Cclsq(1:size(LSQcho,2),1:6,:)=C(LSQcho,1:6,:);
    Cwlsq(1:size(LSQcho,2),1,:)=C(LSQcho,7,:);
    Cccon(1:size(CONcho,2),1:6,:)=C(CONcho,1:6,:);
    Cwcon(1:size(CONcho,2),1,:)=C(CONcho,7,:);

    eCcomp(:,1:6,:)=eC(:,1:6,:);
    eCweak(:,1,:)=eC(:,7,:);
    eCclsq(1:size(LSQcho,2),1:6,:)=eC(LSQcho,1:6,:);
    eCwlsq(1:size(LSQcho,2),1,:)=eC(LSQcho,7,:);
    eCccon(1:size(CONcho,2),1:6,:)=eC(CONcho,1:6,:);
    eCwcon(1:size(CONcho,2),1,:)=eC(CONcho,7,:);
end
```

```
    % Computations of the compensatory torque values...
    % Tcomp is the standard method

    % The "delta computation" using the changes in T. From Matlab if AX=B, then X=A\B
    % A = C*T+GEV & Anew = C*Tnew+GEV --> dA = A-Anew = (C*T+GEV) - (C*Tnew+GEV)
    % dA = (C*T) - (C*Tnew) = C*(T-Tnew) = C*dT
    % dT = C\dA, and since there is no change in A, dA=0
    % and the trivial solution is dT=0.  However, since a portion of dT,
    % Tweak, is fixed at some value, the same algebra in Eq's 1a & 2a
    % above come into play. dT=[dTcomp Tweak]
    % dA(:,:,time) = Ccomp(:,:,time) * dTcomp(:,time) + Cweak(:,:,time) * Tweak(:,time)
    % dTcomp(:,time) = Ccomp(:,:,time) \ (dA(:,:,time) - Cweak(:,:,time) * Tweak(:,time));
    % Again, dA = 0 so
    % dTcomp(:,time) = Ccomp(:,:,time) \ (- Cweak(:,:,time) * Tweak(:,time));

    % The "QR computation" using the QR factorization of Ccomp. From Matlab
    % [Q,R]=qr(C) --> if C is 10x7, then Q is 10x10 and R is 10x7 and Q*R=C
    % Eq.1 A=C*T+GEV yields C*T-(A-GEV)=0. Let A-GEV=b --> C*T-b=0
    % Subbing Q, R for C yields R*T-Q'*b=0. With the Tcomp substitutions,
    % Tqrcomp = Rcomp \ (Qcomp' * (A - Cweak * Tweak - GEV))

    % Using lsqlin constrained linear least squares method.
    % Notation for this is Talsq & Clsq
    %  X=LSQLIN(F,d,A,b,Aeq,beq) solves the least-squares
    %  (with equality constraints) problem:
    %   F*x = d
    %  min  0.5*(NORM(F*x-d)).^2    subject to
    %   x                              A*x <= b and Aeq*x = beq
    % Since there are no inequalities, A & b are [].
    % Setting the Pelvis accelerations as the contraints, we have:
    % x is the vector of torques, T
    % F(:,:,time)==Cclsq & d(:,:,time)==(Alsq(:,:,time)-Cclsq(:,:,time)*Tweak(:,time)-
GEVlsq(:,:,time)).
    % Aeq(:,:,time)==Cccon(:,:,time) & beq(:,:,time)==(Acon(:,:,time)-
Cwcon(:,:,time)*Tweak(:,time)-GEVcon(:,:,time)).

    warning off
    for i=ti
        Tcomp(:,i)=Ccomp(:,:,i)\(A(:,:,i)-Cweak(:,:,i)*Tweak(:,i)-GEV(:,:,i));

        d(:,:,i)=Alsq(:,:,i)-Cwlsq(:,:,i)*Tweak(:,i)-GEVlsq(:,:,i);
        beq(:,:,i)=(Acon(:,:,i)-Cwcon(:,:,i)*Tweak(:,i)-GEVcon(:,:,i));
        Tlsqc(:,i)=lsqlin(Cclsq(:,:,i),d(:,:,i),[],[],Cccon(:,:,i),beq(:,:,i));

        eTcomp(:,i)=eCcomp(:,:,i)\(A(:,:,i)-eCweak(:,:,i)*Tweak(:,i)-eGV(:,:,i));

        ed(:,:,i)=Alsq(:,:,i)-eCwlsq(:,:,i)*Tweak(:,i)-eGVlsq(:,:,i);
        ebeq(:,:,i)=(Acon(:,:,i)-eCwcon(:,:,i)*Tweak(:,i)-eGVcon(:,:,i));
        eTlsqc(:,i)=lsqlin(eCclsq(:,:,i),ed(:,:,i),[],[],eCccon(:,:,i),ebeq(:,:,i));
    end
    warning on

    % Reassembling the complete torque matrix with the compensated values.
    % From C*(T-Tnew) = C*dT  -->  Tdnew = T-dT
    if weak_grp==1 & side==1 % right hip extensors are weak
        Tnew=[Tcomp(1,:)' Tweak' Tcomp(2:6,:)'];
        Tlsqnew=[Tlsqc(1,:)' Tweak' Tlsqc(2:6,:)'];
        eTnew=[eTcomp(1,:)' Tweak' eTcomp(2:6,:)'];
        eTlsqnew=[eTlsqc(1,:)' Tweak' eTlsqc(2:6,:)'];
    elseif weak_grp==1 & side==2 % left hip extensors are weak
        Tnew=[Tcomp(1:2,:)' Tweak' Tcomp(3:6,:)'];
        Tlsqnew=[Tlsqc(1:2,:)' Tweak' Tlsqc(3:6,:)'];
        eTnew=[eTcomp(1:2,:)' Tweak' eTcomp(3:6,:)'];
        eTlsqnew=[eTlsqc(1:2,:)' Tweak' eTlsqc(3:6,:)'];
    elseif weak_grp==2 & side==1 % right knee extensors are weak
        Tnew=[Tcomp(1:3,:)' Tweak' Tcomp(4:6,:)'];
        Tlsqnew=[Tlsqc(1:3,:)' Tweak' Tlsqc(4:6,:)'];
        eTnew=[eTcomp(1:3,:)' Tweak' eTcomp(4:6,:)'];
        eTlsqnew=[eTlsqc(1:3,:)' Tweak' eTlsqc(4:6,:)'];
    elseif weak_grp==2 & side==2 % left knee extensors are weak
        Tnew=[Tcomp(1:4,:)' Tweak' Tcomp(5:6,:)'];
        Tlsqnew=[Tlsqc(1:4,:)' Tweak' Tlsqc(5:6,:)'];
```

```
        eTnew=[eTcomp(1:4,:)' Tweak' eTcomp(5:6,:)'];
        eTlsqnew=[eTlsqc(1:4,:)' Tweak' eTlsqc(5:6,:)'];
    elseif weak_grp==3 & side==1 % right ankle extensors are weak
        Tnew=[Tcomp(1:5,:)' Tweak' Tcomp(6,:)'];
        Tlsqnew=[Tlsqc(1:5,:)' Tweak' Tlsqc(6,:)'];
        eTnew=[eTcomp(1:5,:)' Tweak' eTcomp(6,:)'];
        eTlsqnew=[eTlsqc(1:5,:)' Tweak' eTlsqc(6,:)'];
    elseif weak_grp==3 & side==2 % left ankle extensors are weak
        Tnew=[Tcomp' Tweak'];
        Tlsqnew=[Tlsqc' Tweak'];
        eTnew=[eTcomp' Tweak'];
        eTlsqnew=[eTlsqc' Tweak'];
    end

    % Tac consists the compensated active torques for the ADAMS open-loop
    % co-simulation.  This is based on the following asssumptions:
    % Net joint accelerations & positions have not changed from the non-weakened
    % simulation, and hence Tp has not changed.  This will allow the OL sim to be
    % run with Tp intact.
    Tac(ti,:)=Tnew(ti,:)-Tp(ti,:); % joint torque, extension +ve.
    Talsqc(ti,:)=Tlsqnew(ti,:)-Tp(ti,:); % joint torque, extension +ve.
    Tnew=Tnew';
    Tlsqnew=Tlsqnew';

    eTac(ti,:)=eTnew(ti,:)-Tp(ti,:); % joint torque, extension +ve.
    eTalsqc(ti,:)=eTlsqnew(ti,:)-Tp(ti,:); % joint torque, extension +ve.
    eTnew=eTnew';
    eTlsqnew=eTlsqnew';
end

if 1 % the rest is for plotting & saving the results
    % For error term not included
    figure(1),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(1,ti),'ko-',ti,Tnew(1,ti),'b.-
',ti,Tlsqnew(1,ti),'g.-'),grid, title('Lumbar torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(2),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(2,ti),'ko-',ti,Tnew(2,ti),'b.-
',ti,Tlsqnew(2,ti),'g.-'),grid, title('RHip torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(3),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(3,ti),'ko-',ti,Tnew(3,ti),'b.-
',ti,Tlsqnew(3,ti),'g.-'),grid, title('LHip torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(4),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(4,ti),'ko-',ti,Tnew(4,ti),'b.-
',ti,Tlsqnew(4,ti),'g.-'),grid, title('RKnee torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(5),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(5,ti),'ko-',ti,Tnew(5,ti),'b.-
',ti,Tlsqnew(5,ti),'g.-'),grid, title('LKnee torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(6),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(6,ti),'ko-',ti,Tnew(6,ti),'b.-
',ti,Tlsqnew(6,ti),'g.-'),grid, title('RAnkle torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(7),set(gcf,'Name',name),subplot(2,1,1),plot(ti,T(7,ti),'ko-',ti,Tnew(7,ti),'b.-
',ti,Tlsqnew(7,ti),'g.-'),grid, title('LAnkle torques(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    % For error term included
    figure(1),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(1,ti),'ko-',ti,eTnew(1,ti),'b.-
',ti,eTlsqnew(1,ti),'g.-'),grid, title('Lumbar torques(calculated with error
included)');legend('old','new','LSQLIN');
    figure(2),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(2,ti),'ko-',ti,eTnew(2,ti),'b.-
',ti,eTlsqnew(2,ti),'g.-'),grid, title('RHip torques(calculated with error
included)');legend('old','new','LSQLIN');
    figure(3),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(3,ti),'ko-',ti,eTnew(3,ti),'b.-
',ti,eTlsqnew(3,ti),'g.-'),grid, title('LHip torques(calculated with error
included)');legend('old','new','LSQLIN');
    figure(4),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(4,ti),'ko-',ti,eTnew(4,ti),'b.-
',ti,eTlsqnew(4,ti),'g.-'),grid, title('RKnee torques(calculated with error
included)');legend('old','new','LSQLIN');
    figure(5),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(5,ti),'ko-',ti,eTnew(5,ti),'b.-
',ti,eTlsqnew(5,ti),'g.-'),grid, title('LKnee torques(calculated with error
included)');legend('old','new','LSQLIN');
    figure(6),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(6,ti),'ko-',ti,eTnew(6,ti),'b.-
',ti,eTlsqnew(6,ti),'g.-'),grid, title('RAnkle torques(calculated with error
included)');legend('old','new','LSQLIN');
```

```
    figure(7),set(gcf,'Name',name),subplot(2,1,2),plot(ti,T(7,ti),'ko-',ti,eTnew(7,ti),'b.-
',ti,eTlsqnew(7,ti),'g.-'),grid, title('LAnkle torques(calculated with error
included)');legend('old','new','LSQLIN');

    % Anew is the recalculated accelerations using the compensated joint torques and
    % the original positional inertia matrix, and is plotted against the original
    % accelerations.
    for i=ti
        % For error term not included
        Anew(:,:,i)=C(:,:,i)*Tnew(:,i)+GEV(:,:,i);
        Alsqnew(:,:,i)=C(:,:,i)*Tlsqnew(:,i)+GEV(:,:,i);
        % For error term included
        eAnew(:,:,i)=eC(:,:,i)*eTnew(:,i)+eGV(:,:,i);
        eAlsqnew(:,:,i)=eC(:,:,i)*eTlsqnew(:,i)+eGV(:,:,i);
    end

    % For error term not included
    figure(11),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(1,ti),'ko-',ti,Anew(1,ti),'b.-
',ti,Alsqnew(1,ti),'g.-'),grid, title('Lumbar acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(12),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(2,ti),'ko-',ti,Anew(2,ti),'b.-
',ti,Alsqnew(2,ti),'g.-'),grid, title('RHip acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(13),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(3,ti),'ko-',ti,Anew(3,ti),'b.-
',ti,Alsqnew(3,ti),'g.-'),grid, title('RKnee acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(14),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(4,ti),'ko-',ti,Anew(4,ti),'b.-
',ti,Alsqnew(4,ti),'g.-'),grid, title('RAnkle acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(15),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(5,ti),'ko-',ti,Anew(5,ti),'b.-
',ti,Alsqnew(5,ti),'g.-'),grid, title('LHip acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(16),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(6,ti),'ko-',ti,Anew(6,ti),'b.-
',ti,Alsqnew(6,ti),'g.-'),grid, title('LKnee acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(17),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(7,ti),'ko-',ti,Anew(7,ti),'b.-
',ti,Alsqnew(7,ti),'g.-'),grid, title('LAnkle acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(18),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(8,ti),'ko-',ti,Anew(8,ti),'b.-
',ti,Alsqnew(8,ti),'g.-'),grid, title('Pelvis forward acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(19),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(9,ti),'ko-',ti,Anew(9,ti),'b.-
',ti,Alsqnew(9,ti),'g.-'),grid, title('Pelvis vertical acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    figure(20),set(gcf,'Name',name),subplot(2,1,1),plot(ti,A(10,ti),'ko-',ti,Anew(10,ti),'b.-
',ti,Alsqnew(10,ti),'g.-'),grid, title('Pelvis rotation acc(calculated with error as a seperate
term)');legend('old','new','LSQLIN');
    % For error term included
    figure(11),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(1,ti),'ko-',ti,eAnew(1,ti),'b.-
',ti,eAlsqnew(1,ti),'g.-'),grid, title('Lumbar acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(12),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(2,ti),'ko-',ti,eAnew(2,ti),'b.-
',ti,eAlsqnew(2,ti),'g.-'),grid, title('RHip acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(13),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(3,ti),'ko-',ti,eAnew(3,ti),'b.-
',ti,eAlsqnew(3,ti),'g.-'),grid, title('RKnee acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(14),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(4,ti),'ko-',ti,eAnew(4,ti),'b.-
',ti,eAlsqnew(4,ti),'g.-'),grid, title('RAnkle acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(15),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(5,ti),'ko-',ti,eAnew(5,ti),'b.-
',ti,eAlsqnew(5,ti),'g.-'),grid, title('LHip acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(16),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(6,ti),'ko-',ti,eAnew(6,ti),'b.-
',ti,eAlsqnew(6,ti),'g.-'),grid, title('LKnee acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(17),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(7,ti),'ko-',ti,eAnew(7,ti),'b.-
',ti,eAlsqnew(7,ti),'g.-'),grid, title('LAnkle acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(18),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(8,ti),'ko-',ti,eAnew(8,ti),'b.-
',ti,eAlsqnew(8,ti),'g.-'),grid, title('Pelvis forward acc(calculated with error
included)');legend('old','new','LSQLIN');
```

```
    figure(19),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(9,ti),'ko-',ti,eAnew(9,ti),'b.-
',ti,eAlsqnew(9,ti),'g.-'),grid, title('Pelvis vertical acc(calculated with error
included)');legend('old','new','LSQLIN');
    figure(20),set(gcf,'Name',name),subplot(2,1,2),plot(ti,A(10,ti),'ko-',ti,eAnew(10,ti),'b.-
',ti,eAlsqnew(10,ti),'g.-'),grid, title('Pelvis rotation acc(calculated with error
included)');legend('old','new','LSQLIN');

    for i=ti
        if 0
            % For error term not included
            LUMacc_new(i,3)=Anew(1,1,i);
            RHacc_new(i,3)=Anew(2,1,i);
            RKacc_new(i,3)=Anew(3,1,i);
            RAacc_new(i,3)=Anew(4,1,i);
            LHacc_new(i,3)=Anew(5,1,i);
            LKacc_new(i,3)=Anew(6,1,i);
            LAacc_new(i,3)=Anew(7,1,i);
            PELVISacc_new(i,1)=Anew(8,1,i);
            PELVISacc_new(i,2)=Anew(9,1,i);
            PELVISacc_new(i,3)=Anew(10,1,i);
        end

        % For error term included
        LUMacc_new(i,3)=eAnew(1,1,i);
        RHacc_new(i,3)=eAnew(2,1,i);
        RKacc_new(i,3)=eAnew(3,1,i);
        RAacc_new(i,3)=eAnew(4,1,i);
        LHacc_new(i,3)=eAnew(5,1,i);
        LKacc_new(i,3)=eAnew(6,1,i);
        LAacc_new(i,3)=eAnew(7,1,i);
        PELVISacc_new(i,1)=eAnew(8,1,i);
        PELVISacc_new(i,2)=eAnew(9,1,i);
        PELVISacc_new(i,3)=eAnew(10,1,i);
    end

    if 0
        save(['TacAllData_',prefix,'_',wkname]);

save(['TacOLdata_',prefix,'_',wkname],'Opos*','pos*','T*','VV','time','prefix','tstart','tstop');
    end
    save(['comp_',prefix,'_',wkname],'*new','wkname');
end
```